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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so.

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ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 15 double-spaced typed pages. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

Manuscripts for and correspondence about this publication should be sent to:

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IN THIS ISSUE

VIRUS

HORACE V. WESTER and EDWARD W. JYLKKA have succeeded in transmitting elm scorch (foliar symptoms of which are similar to those of Pierce's disease of grape) from diseased to healthy trees by both chip bud and scion grafts, indicating a virus as the cause of the disease, page 519.

HARRY P. KARLE and GEORGE NYLAND call attention to an almond orchard in California in which the trees were severely infected by the yellow bud mosaic virus in 1958, page 520.

Results of sorghum inoculation tests with the sugarcane mosaic virus, conducted by JACK L. DEAN and OTTO H. COLEMAN at the U. S. Sugar Crops Field Station, Meridian, Mississippi, have shown that while many sorghum varieties give the characteristic well known mottle symptoms, some varieties give a necrotic reaction, while still others appear to possess varying degrees of resistance, page 522.

L. G. WEATHERS and E. C. CALAVAN report the results of cachexia-xyloporosis virus indexing tests of various commercially-grown California lemon varieties and selections, page 528.

A fast, economical index method of testing for Kwanzan-systemic virus, using greenhouse facilities, is described by HAROLD E. WILLIAMS and H. K. WAGNON, page 534.

FUNGICIDES

Cyprex was by far the best of eight fungicides tested by R. E. INMAN and J. L. WEIHING in a Nebraska nursery for control of shothole disease of chokecherry, page 536.

In New Hampshire, AVERY E. RICH and M. C. RICHARDS have demonstrated through numerous trials that both time and space may be saved in the evaluation of fungicides for control of apple scab if apple seedlings rather than budded trees are used for inoculation experiments, page 540.

Based on a 3-year study at the University of Delaware Research Farm, D. F. CROSSAN discusses the efficacy of several chemical treatments applied to cut seed pieces of the Cobbler variety of potato for the prevention of bacterial and Fusarium decay under different environmental and soil conditions, page 543.

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HUEY I. BORDER's investigations into the physiological effect on the plant of several chemicals used as seed treatments to control pathogens borne on the surface of cabbage seed revealed that, of the materials tested, only the antibiotic streptomycin (in strengths as low as 25 ppm) caused a purplish discoloration of seedlings that led to early death of the plants, page 549.

Two experimental compounds, Acti-dione-S and D-113, show promise of equalling or bettering the performance of zineb for control of asparagus rust, according to the field screening tests of HARRY H. MURAKISHI, page 552.

Results of comparative tests in Canada, by BJORN PETURSON and F. R. FORSYTH, indicate that application of certain cereal rust fungicides in low volumes of water is just as effective, if not more so, as application in high volumes of water, page 556.

D. C. ERWIN, H. T. REYNOLDS, and M. J. GARBER present results of their 1958 cotton seed treatment tests in California, designed to compare the efficacy of Thimet-captan with other Thimet-fungicide combinations, and to observe the effect of Thimet on germination of cotton in the field, page 558.

According to the experiments of R. B. VALDEZ, A. N. PORDESIMO, and F. T. ORILLO the fungicides Parzate in combination with such good commercial stickers as Goodrite p.e.p.s. or Triton B-1956 may be superior to Bordeaux mixture for control of coffee rust under the conditions of heavy precipitation and high relative humidity that prevail in the Philippines, page 562.

As part of a study of possible formulations of antiseptic tree would paints, CURTIS MAY and JOHN G. PALMER experimented with fungicide-asphalt mixtures as inhibitors in vitro of the fungus causing a fatal disease of the London planetree, and found that five of the eight

fungicides tried prevented fungal growth, page 565.

FIELD RESISTANCE IN CROP VARIETIES

E. H. VARNEY, J. N. MOORE, and D. H. SCOTT summarize the findings of their preliminary field screening of 50 strawberry varieties or selections for resistance to Verticillium wilt in New Jersey, page 567.

N. N. WINSTEAD, M. J. GOODE, and W. S. BARHAM have shed a little more light on the nature of resistance in watermelon to anthracnose by their recent screening of 86 available varieties for resistance to races 1, 2, and 3 and their evaluation of about 350 Plant Introductions for resistance to race 2, page 570.

In an attempt to discover additional varieties of rye, besides the varieties Gator and Explorer, with resistance to leaf rust, DARRELL D. MOREY was unable to find any foreign or domestic types that approached Gator in resistance, with the exception of lines selected from Gator, page 578.

MISCELLANEOUS

In California, IVAN J. THOMASON obtained good control to a depth of 3 feet of root-knot nematodes and the Fusarium wilt organism with methyl bromide injected into the soil at dosages of 200 and 300 pounds per acre by chisel applicator and then covered with tarps -- further evidence of the advisability of utilizing this fumigant economically for soil treatment on large areas, page 580.

P. M. HALISKY, R. H. GARBER, and W. C. SCHNATHORST made a study of the importance of environmental factors upon Verticillium hadromycosis of cotton and found that the influence of soil temperature on wilt development is directly related to the effect of temperature on growth and activity of the pathogen, page 584.

According to investigations conducted by W. H. GILLESPIE and R. P. TRUE in West Virginia, local spread of oak wilt in five northeastern counties of the State is favored by shallow soils, availability of "compatible" oaks, and number of dead and currently wilting trees at the infection center when found and treated, page 588.

Brief Notes on Plant Disease Occurrence, page 594: A new host for the larch dwarfmistletoe, by DONALD P. GRAHAM. A latent virus of hops detected by cucumber inoculation, by P. R. FRIDLUND. New records of plant diseases in New Mexico, by C. H. HSI.

Announcement, page 595.

Correction, page 595.

March Weather, page 596.

ELM SCORCH, GRAFT TRANSMISSIBLE VIRUS OF AMERICAN ELM

Horace V. Wester and Edward W. Jylkka[1]

Elm scorch, a foliar necrosis, is associated with gradual crown deterioration and failure of American elm, Ulmus americana. This disease has become serious in the Washington, D. C. area during the past decade. It also appears to be widespread in the southeastern States according to exploratory surveys conducted in 1957 and 1958.

A series of transmission studies were conducted from 1951 to 1958 on 3- and 4-year-old American elms growing under nursery conditions. Inoculations were by chip buds[2], scions, and bark patches. Cultures made from elm scorch inoculum and from trees developing disease symptoms yielded only a few bacteria, which were considered contaminants.

From a total of 213 chip bud-inoculated elms, where the inoculum was from diseased elms, 18.3 percent developed symptoms of the disease in the first growing season following inoculation and 21.1 percent in the second. During the same period no symptoms of the disease developed on 67 elms inoculated with chip buds from healthy elms. In 39 and 17 scion inoculated elms, where inoculum was respectively from diseased and healthy elm sources, 18 percent of the elms inoculated with diseased inoculum developed elm scorch symptoms, while no disease symptoms developed on the trees inoculated with healthy inoculum. Inoculum from diseased and healthy trees was applied by bark patches to 170 and 108 elms, respectively. In both groups only one case of elm scorch developed; these infections probably originated from natural causes.

The results of these transmission tests show that elm scorch symptoms are transmissible by chip bud and scion inoculum, but not by bark patches. Transmission reported from chip bud and scion inoculum is considered significant and indicates a virus to be the cause of this disease. Moreover, as transmission resulted only from inoculum containing xylem tissue as in chip buds and scions, and not from strictly phloem inoculum as in bark patches, it seems reasonable to conclude that the virus develops in the xylem and not in the phloem.

The foliar symptoms of elm scorch are somewhat similar to Pierce's disease of grape (1) which is also caused by a virus (Morsus suffodiens Holmes) that develops in the xylem (2). This virus may prove to be the cause of elm scorch of American elm.

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1. HEWITT, W. B., N. W. FRAZIER, H. E. JACOBS, and J. H. FREITAG. 1942. Pierce's disease of grapevines. California Agr. Exp. Sta. Circ. 353.
2. HOUSTON, B. R., K. ESAU, and Wm. B. HEWITT. 1947. The mode of vector feeding and the tissues involved in the transmission of Pierce's disease virus in grape and alfalfa. Phytopathology 37: 247-253.

NATIONAL CAPITAL PARKS, NATIONAL PARK SERVICE, UNITED STATES DEPARTMENT OF INTERIOR

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[1] Plant Pathologists, National Capital Parks, National Park Service, United States Department of Interior, Washington, D. C.

[2] Chip bud refers to shield bud with adhering wood chip.

YELLOW BUD MOSAIC VIRUS IN ALMOND

Harry P. Karle and George Nyland

In May 1958 our attention was called to a severely diseased almond orchard near Winters, California. This orchard was one of the places where yellow bud mosaic was first discovered in California (1), but the crop involved then was peaches. Almonds replaced the peaches in about 1943. Symptoms on almond were typical of the yellow bud mosaic disease, but much more severe and damaging than previously reported for almonds (2). The orchard was planted mainly to the variety Texas, with Nonpareil as the primary pollinizer variety. Some trees of Ne Plus also were present. It was thought that a report would be of interest because of the extent of damage to the trees and the distribution of disease in the trees.

The almond orchard contained 1241 trees, with 1108 (89 percent) affected. Symptoms were abundant throughout the Texas variety, extending to the uppermost portions of many trees (Fig. 1, A). Chlorotic spotting, vein clearing, and the vein feathering symptom were seen occasionally on some leaves. The symptoms consisted mainly of sparse foliation, tufting, and lack of terminal growth. Some affected trees showed considerable spur and lateral bud failure, which gave the trees a willowy and open appearance. Leaves that were present were smaller than normal (Fig. 1, B) and occurred as terminal tufts (Fig. 1, C). Frequently, one or more normal shoots were produced from otherwise completely diseased branches (Fig. 1, A). Some severely affected branches showed evidence of dieback, with vigorous, normal-appearing current season shoots near their bases. Sucker growth from the trunks usually showed rosette-type growth (Fig. 1, D). Yellow bud mosaic virus was readily recovered by juice inoculation to herbaceous hosts from leaves on these shoots.

The crop in this orchard varied considerably among diseased and normal-appearing trees. Reduction in yield was directly proportional to severity of symptoms. Although many of the fruits on affected limbs were larger than those on unaffected limbs, they were extremely roughened and wrinkled, with hulls abnormally thickened (Fig. 1, E). This symptom was mentioned in the original report on yellow bud mosaic, but was not definitely attributed to this virus (1). Other viruses are known to cause roughened fruits of Texas almond, for example, cherry rugose mosaic and "almond mosaic."

The variety Texas was much more severely affected than either Nonpareil or Ne Plus. In the latter varieties symptoms were seldom evident above the main trunk or bases of the main scaffold branches, whereas in Texas, symptoms extended to the very tops of the trees. Observations in this orchard indicate that planting Texas almond is not advisable in soil known to be infested with yellow bud mosaic virus.

The original planting of almond trees in one section of the orchard had been killed by Armillaria mellea. In that section replants of Marianna 26-24 were used as a rootstock for almond. None of those trees showed symptoms of yellow bud mosaic. Of the tree fruit rootstocks, only peach and almond have become infected when planted in infested soil, as far as now known.

Literature Cited

1. THOMAS, H. EARL, and T. E. RAWLINS. 1939. Some mosaic diseases of Prunus species. Hilgardia 12: 623-644.
2. THOMAS, H. EARL, and T. E. RAWLINS. 1951. Yellow bud mosaic. In Virus diseases and other disorders with virus-like symptoms of stone fruits in North America. United States Department of Agriculture Handbook 10, pp. 53-55.

FIGURE 1. A -- Texas almond showing dieback, sparse foliation, tufting and lack of terminal growth. Arrows point to some normal shoots on otherwise completely diseased branches.

B -- Normal appearing branch of Texas almond.

C --YBMV infected limb of Texas almond showing lack of growth, terminal tufting and fruits with excessively wrinkled hulls.

D -- Suckers on a main scaffold limb of a tree of Texas almond showing rosette-type growth.

E -- Texas almond. Left, normal fruit; Right, fruit from diseased branches.

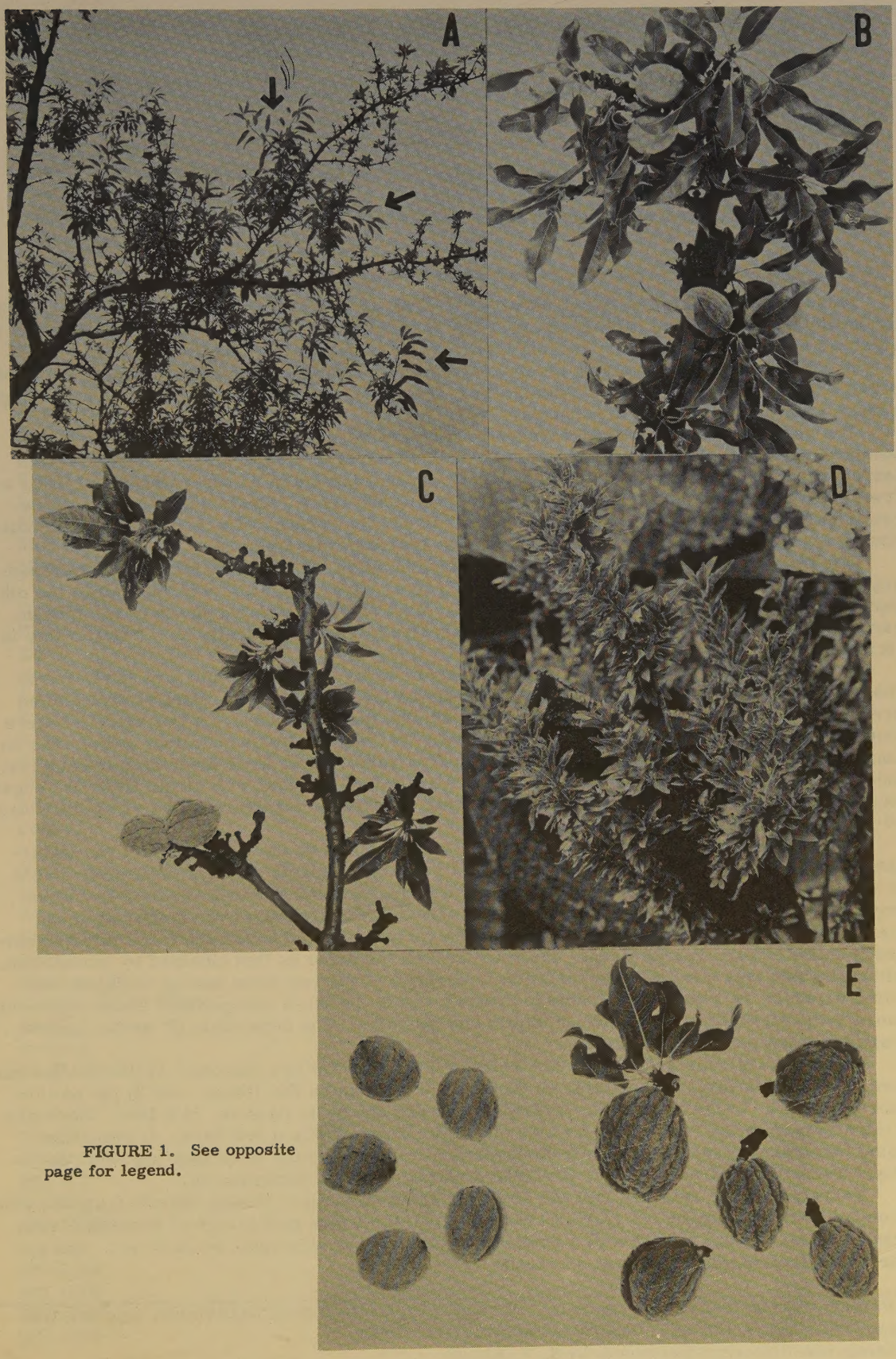

FIGURE 1. See opposite page for legend.

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NECROTIC AND RESISTANT REACTIONS TO THE SUGARCANE MOSAIC VIRUS, IN SORGHUM

Jack L. Dean and Otto H. Coleman[1]

Abstract

A severe necrosis of sorghum leaves was found to be a reaction of particular varieties to infection by the sugarcane mosaic virus and resistance to sugarcane mosaic was found in sorghum.

Most sorghum varieties (Sorghum vulgare) react to infection with sugarcane mosaic virus by developing a light- and dark-green mottling of the leaves. This well known reaction will be referred to herein as "ordinary mosaic mottling" or simply as the "mottle reaction."

A different reaction involving the development of necrotic leaf patterns has been observed at the United States Sugar Crops Field Station, Meridian, Mississippi, and resistance to sugarcane mosaic has been found in sorghum.

The necrotic reaction was discovered in attempts to establish the nature of what was thought to be a new disease of sorghum. Several attempts to isolate fungi or bacteria from leaf lesions yielded either no cultures, or cultures that proved to be non-pathogenic. Only a few affected plants had been observed before 1954, when two F_1 plants displaying gaudy, necrotic foliage patterns were found in the field. In addition to necrosis, ordinary mosaic mottling was present in two or three of the youngest leaves of these plants.

The parents of each of these F_1 plants were planted in the field and later inoculated with sugarcane mosaic virus. One parent of each F_1 plant gave the mottle reaction, while the other parent in each case gave the necrotic reaction. Most of the inoculated plants of both parent varieties that reacted by developing necrosis eventually died, apparently as a result of the infection.

In order to determine whether relatively few or many varieties would give the necrotic reaction, a series of inoculation tests was conducted in 1955 and 1956. Each test contained previously inoculated varieties of known reaction plus some new ones. A few varieties were inoculated at various stages of growth in the field, but most of the inoculations were made on small plants in the greenhouse. Ten to 15 plants of each variety were inoculated in each test. Inoculum consisted of expressed sap of Co. 290 sugarcane infected with strain B[2] of the sugarcane mosaic virus. Inoculation was accomplished by rubbing a mixture of the virus juice and 80- to 100-mesh sand on the leaves.

The reactions of the varieties inoculated were classified as mottle, necrotic, or resistant, as indicated in Table 1. The reaction of a particular variety appears to be quite stable. In no case did a variety give a necrotic reaction in one test and a mottle reaction in another, regardless of whether young plants were inoculated in the greenhouse or relatively large plants were inoculated in the field. Many of the varieties tested have been found infected naturally in field plots; in every case, the reaction was the same as that obtained by inoculation. Although there was considerable variation in severity of the reactions among varieties classified together, no attempt was made to classify within the three categories. There were only two or three borderline cases between mottle and necrotic. The separation of mottle and resistant was not quite as definite.

The varieties considered resistant were so classified for two reasons: 1) the mottled pattern developed at least a week later on these varieties than on the others, and 2) the pattern was very mild. In the case of the variety Wiley, and one of its parents, MN 2046, there was a third consideration, the disappearance of all symptoms within a few days. It should be noted that ordinary mosaic mottling disappears from all varieties when they approach maturity. The symptoms in Wiley and MN 2046 disappeared from young plants.

There was some evidence that the resistant group is further distinguishable from the other groups in that, in a given test, inoculation resulted in a lower percentage of infected plants. Since a small number of plants of a particular variety were inoculated in each test, this apparent distinction may have been spurious.

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[1] Pathologist and Research Agronomist, respectively, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[2] Strain identifications were made by E. V. Abbott.

Table 1. Reactions of sorghum varieties or selections to sugarcane mosaic virus.

Variety or selection	Identification number	Number of tests	Reaction
Albaugh	F.C. 16158[a]	1	Mottle
Ames Amber	F.C. 13575	1	Mottle
Atlas	F.C. 9112	5	Necrotic
Chinese Amber	F.C. 8728	1	Mottle
Clubhead	F.C. 8708	1	Mottle
Colman (M)	F.C. 16159	1	Mottle
Cowper	F.C. 13615	1	Mottle
Early Folger	F.C. 9097	1	Mottle
Early Folger	F.C. 16154	1	Mottle
Early Sumac	F.C. 6611	1	Mottle
Gooseneck	F.C. 16165	2	Mottle
Honey	F.C. 6605	1	Mottle
Honey Drip	F.C. 16164	1	Mottle
Indiana Amber	F.C. 16152	2	Necrotic
Jones	F.C. 16163	1	Mottle
Kansas Orange	F.C. 9108	1	Mottle
Leoti	F.C. 6610	2	Necrotic
Mazo Amber	F.C. 16153	1	Mottle
McLean	F.C. 13439	2	Necrotic
Minnesota Amber	F.C. 16151	1	Mottle
Orange	S.A. 20[b]	1	Necrotic
Planter	F.C. 16156	5	Necrotic
Rex	F.C. 16185	2	Mottle
Red Amber	F.C. 17548	1	Mottle
Rox Orange	F.C. 13640	1	Mottle
Saccaline	F.P.I. 48191[c]	1	Mottle
Sugar Drip	F.C. 16161	1	Mottle
Sourless	F.C. 9111	1	Mottle
Straightneck	F.C. 13490	1	Mottle
Silver Top	F.C. 16162	1	Necrotic
Sumac	F.P.I. 35038	1	Mottle
Texas Seeded Ribbon	F.C. 16167	1	Mottle
Waconia Amber	F.C. 16205	1	Mottle
White African	F.C. 6604	2	Mottle
Tracy		5	Mottle
Collier		1	Mottle
Colman (Y)		1	Mottle
Georgia Blue Ribbon		1	Necrotic
Hodo		1	Mottle
Iceberg		1	Necrotic
C. P. Special		3	Necrotic
Hegari	S.P.I. 22326[d]	1	Mottle
Norkan		1	Necrotic
Western Blackhull		1	Necrotic
MN 876	P.E.I. 152747[e]	3	Resistant
MN 960	P.E.I. 152857	2	Resistant
MN 1032[f]	P.E.I. 15243	3	Resistant
Sart	P.E.I. 152945	6	Mottle
MN 1049	P.E.I. 152960	3	Resistant
MN 1052	P.E.I. 152963	3	Necrotic
MN 1054	P.E.I. 152965	2	Resistant
MN 1056	P.E.I. 152967	4	Necrotic

Table 1. Continued.

Variety or selection	Identification number	Number of tests	Reaction
MN 1058 (Brown)	P.E.I. 152969	2	Resistant
MN 1060	P.E.I. 152971	1	Mottle
Williams		2	Necrotic
MN 1996	P.E.I. 155780	2	Mottle
MN 2029	P.E.I. 155803	2	Mottle
MN 2046	P.E.I. 155819	2	Resistant
MN 2648	P.E.I. 168502	2	Mottle
African Kafir	F.P.I. 52606	1	Necrotic
Beaver		1	Necrotic
Plainsman		1	Necrotic
Pythium-Resistant Quadroon		2	Mottle
Juba Kafir	F.P.I. 51609	1	Mottle
Brown Kaoliang	F.P.I. 66384	1	Mottle
MN 2860	P.E.I. 173971	1	Necrotic
Wiley		5	Resistant
Mer. 51-2[g]		1	Mottle
Crystal Drip		1	Mottle
Tennessee Red Top		1	Mottle
Williams		2	Necrotic
Orange-Centered Iceberg		1	Necrotic

[a] F.C. = Forage Crops accession number.
[b] S.A. = Sorghum accession number (Chillicothe, Texas).
[c] F.P.I. = Plant Introduction accession number.
[d] S.P.I. = Plant Introduction accession number.
[e] P.E.I. = Plant Introduction accession number.
[f] MN = "Meridian" accession number (Sugarcane and Sweet Sorghum Section).
[g] Mer. = Meridan (Mississippi) selection number.

Resistance to mosaic seems to be less common in sorghum than in sugarcane. Among the varieties tested, only Wiley and MN 2046 appear to have a degree of resistance approaching that of the more resistant commercial varieties of sugarcane.

A healthy leaf, a leaf showing a mottled pattern, and leaves showing some of the necrotic patterns that have been observed are shown in Figures 1-5. The patterns involve stripes, spots, zonate spots, and combinations of these. There may have been some tendency for a particular pattern to be a genetic expression of a particular variety, but different patterns were observed on different parts of a single plant and no serious effort was made to clarify this point.

Necrotic patterns have been observed to develop in three fairly distinct ways. In the first case, the young, unfolding leaves exhibit a severe mottled pattern. Within a few hours the chlorotic areas became necrotic at their centers. The necrotic areas may then remain surrounded by a chlorotic halo for several days.

In a second case, necrotic areas are already present when the young leaves unfold. The tissues separating the necrotic areas show ordinary mosaic mottling.

With both of the foregoing reactions, ordinary mosaic mottling is present as one aspect of infection until the plants approach maturity when only necrosis remains.

In a third case, mottling never appears. The young unfolding leaves appear completely normal for a few hours. Then quite suddenly there is an apparent dehydration of leaf tissue in streaks. The streaks quickly become necrotic. This reaction has been observed only in young plants in the greenhouse.

Necrosis may occur on leaf sheaths as well as on blades. Frequently long necrotic stripes are continuous from blade to sheath.

The necrotic reaction in no way resembles a local-lesion reaction. The infection is

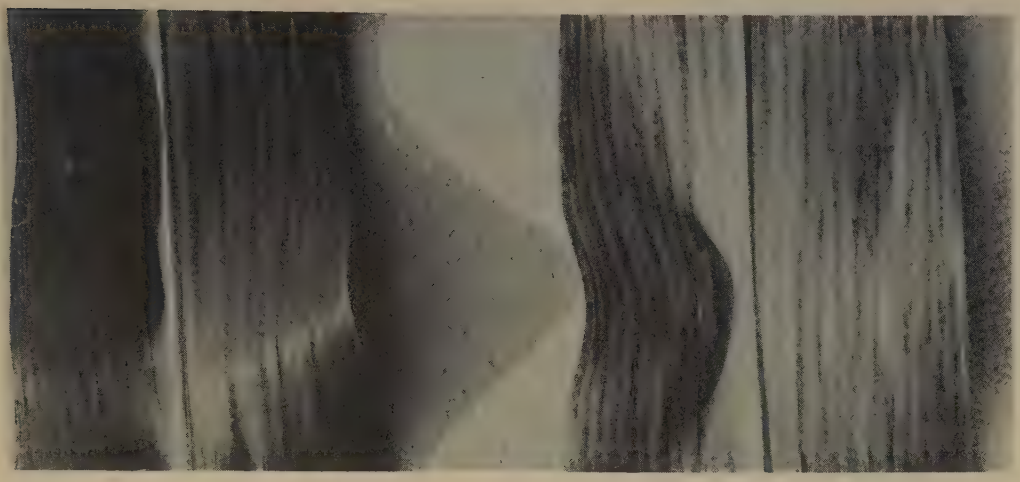

FIGURE 1. Left -- healthy leaf. Right -- leaf showing ordinary mosaic mottling.

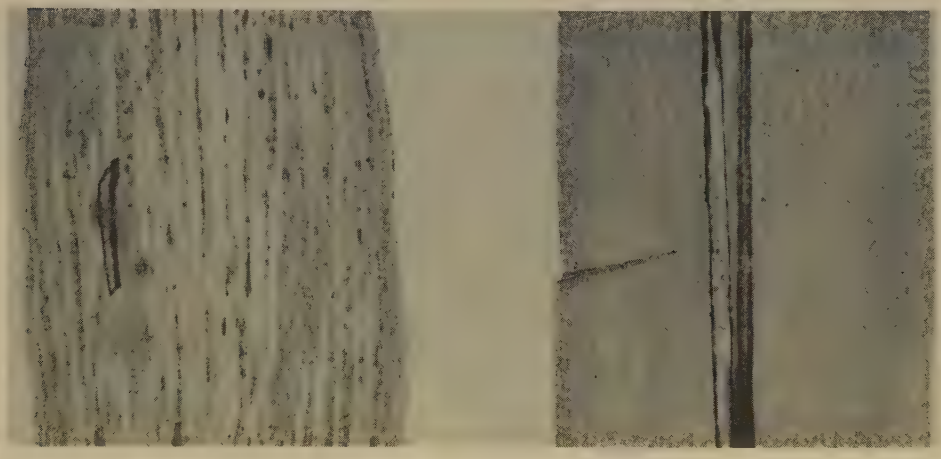

FIGURE 2. Left -- leaf showing a severe, mottled pattern which is becoming necrotic. (Conspicuous elongate spot in left half of blade is a leafhopper egg packet.) Right -- leaf showing a single necrotic stripe as the only evidence of mosaic.

FIGURE 3. Left -- leaf showing necrotic pattern that is a mixture of striping and spotting. Right -- leaf showing streaked, necrotic pattern.

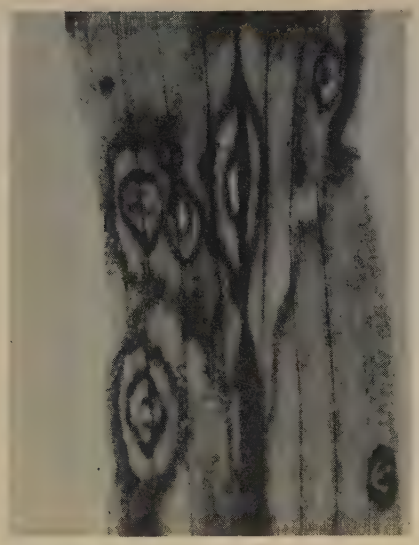

FIGURE 4 (Left). Leaf from small, greenhouse-grown plant showing necrotic pattern in the form of zonate spots.

FIGURE 5 (Below). Sorghum plant showing severe mottling becoming necrotic on young leaves and severe necrosis on older leaves.

always systemic, with necrosis appearing only on leaves unfolded after inoculation.

The more severe of the necrotic patterns are sufficiently injurious, soon after inoculation, to retard or stop growth. Large plants growing in the field may survive and mature seed, or may die prematurely. Plants of a variety that develops a severe necrotic pattern frequently die within 2 weeks after inoculation if they are inoculated during the first week after emergence.

The necrotic reaction of sorghum to sugarcane mosaic virus is probably what has been described in Italy under the name "red streak." According to Lovisolo[3], this disease was described by G. Goidanich in 1939 and studied by E. Corberi in 1940. Goidanich evidently considered the disease a virosis, whereas Corberi considered the evidence inconclusive. Lovisolo concluded that the disease was definitely a virosis and that it was probably caused by a strain of the sugarcane mosaic virus.

Since a wide range in type of reactions to mosaic was found among sorghum varieties, it seemed possible that a set of sorghum differentials could be found to aid in the identification of sugarcane mosaic strains. This possibility was checked by E. V. Abbott at the United States Sugarcane Field Station, Houma, Louisiana. He used strains A, B, and D of the virus on 18 varieties of sorghum selected for the range in type of reaction that they exhibited when they were infected by strain B at Meridian. Dr. Abbott reported (in personal correspondence) that although there was a difference in symptom pattern produced by mosaic on the different varieties, there was no important difference in symptom pattern between strains on any one variety on which infection with more than one strain was obtained.

A similar test was conducted at Meridian with 22 sorghum varieties including some, but not all, of the varieties used by Dr. Abbott, and with strains B and D of the sugarcane mosaic virus. In this test, also, there were wide differences in reaction among varieties, but no apparent differences between strains on any one variety.

Apparently, sorghum is not a promising source of hosts for the differentiation of strains of the sugarcane mosaic virus.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, MERIDIAN, MISSISSIPPI AND THE MISSISSIPPI AGRICULTURAL EXPERIMENT STATION

[3] Lovisolo, Osvaldo. 1956. Contributo sperimental alla conoscenza ed alla determinazione del virus agente dell'arrossamento striato del sorgo e di un mosaico del mais. Bol. della Staz. di Pat. Veg. Anno XIV, ser. 3: 261-321.

THE OCCURRENCE OF CACHEXIA AND XYLOPOROSIS IN CALIFORNIA LEMON VARIETIES, WITH PARTICULAR REFERENCE TO THE OLD-LINE EUREKA LEMON

L. G. Weathers and E. C. Calavan[1]

Abstract

Cachexia-xyloporosis type symptoms have developed on Tien Chieh mandarin seedlings graft-inoculated with tissue from two Lisbon lemon selections (Fritz and Wohlford) neither of which are important commercial selections. Neither cachexia nor xyloporosis symptoms appeared in any indicator plants grafted with scions from other lemon sources. Palestine sweet lime plants grafted with scions from lemon trees known to be infected with exocortis virus developed shallow, elongated cracks in the outer bark of the trunks but no xyloporotic wood pitting. The low incidence of infection shown by these results indicates that the virus or viruses of cachexia and xyloporosis are not widespread in commercial varieties of lemons in California.

Following the discovery of cachexia in Orlando tangelo (Citrus paradisi X C. reticulata) in 1950 by Childs (4) several workers (1, 6, 7, 8, 9) have demonstrated the widespread occurrence of cachexia in a large number of citrus species, varieties, and hybrids throughout the citrus areas of the world. Further investigations by Childs (5) led to the suggestion that cachexia and xyloporosis, described by Reichert and Perlberger (10) in 1934, are caused by the same virus. Calavan and Weathers (3) have reported the finding of a growth-retarding virus in old-line Eureka lemons (C. limon), presently suspected to be the virus of exocortis. They suggest the possibility that this virus is partly responsible for the poor vigor of old-line Eureka lemon trees, and further that it also may be a causal factor in the development of shell bark in many lemon trees. The discovery of the growth-retarding virus in old-line Eureka lemons in 1954 (2) and the fact that the virus of cachexia and/or xyloporosis has been found in many varieties of citrus throughout the world led to studies during 1954-1958 to determine what viruses are present in lemon trees in California.

This paper reports the results of cachexia-xyloporosis virus indexing tests of various commercially-grown California lemon varieties and selections.

INDEXING PROCEDURE

Scions from lemon source trees were grafted into seedlings of one or more of the various susceptible varieties and hybrids of mandarin (C. reticulata) to index for cachexia and into Palestine sweet lime (C. aurantifolia) to index for xyloporosis, since it has not been established with certainty that these two diseases are caused by the same virus. Test plants were selected for uniformity and grown singly in pots in the greenhouse. When the plants were 10 to 14 inches tall they were graft-inoculated with scions from a field tree selected for testing, care being taken that each seedling received scions from only one source. Because the availability of the virus-indexing material varied from season to season many of the lemon trees were tested on only one or two indicator plants while others were tested on several indicator plants. In some of the tests no sprouts were allowed to grow from the inserted scions; in others the inserted scion was forced into growth to form the top of the tree.

Ten to 12 months after inoculation trees were removed from the pots and transplanted into field plots in Riverside and Santa Barbara to await development of symptoms. In order to expose any discoloration or abnormality that might occur a thin section of bark was removed

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periodically near the vicinity of the inserted scion or across the bud-union of the trees on which the scion was forced into growth.

RESULTS

Of 53 old-line Eureka lemon trees, representing 14 selections, tested on various cachexia-susceptible tangelo and mandarin varieties none has caused symptoms of cachexia after 3 to 4 years (Table 1). Scions of nine old-line Eureka lemon trees (two selections) failed to induce symptoms of xyloporosis when grafted into Palestine sweet lime plants. Neither cachexia nor xyloporosis symptoms appeared in any indicator plants grafted with scions from nucellar-line Eureka lemons.

Of the 12 Lisbon lemon source trees (nine selections) tested on virus indicator plants, two showed symptoms suggestive of cachexia and xyloporosis (Table 1). A xyloporotic type of bark pegging with brownish discoloration at the tips coinciding with pitting in the wood developed in Tien Chieh mandarin seedlings inoculated with scions from Wohlford and Fritz old-line Lisbon lemons (Fig. 1). However, scions of these same source trees when inserted into Sunshine tangelo have not induced symptoms of cachexia after 3 years. Neither cachexia nor xyloporosis symptoms developed in virus-index plants grafted with scions from other Lisbon lemon sources or miscellaneous lemon varieties tested.

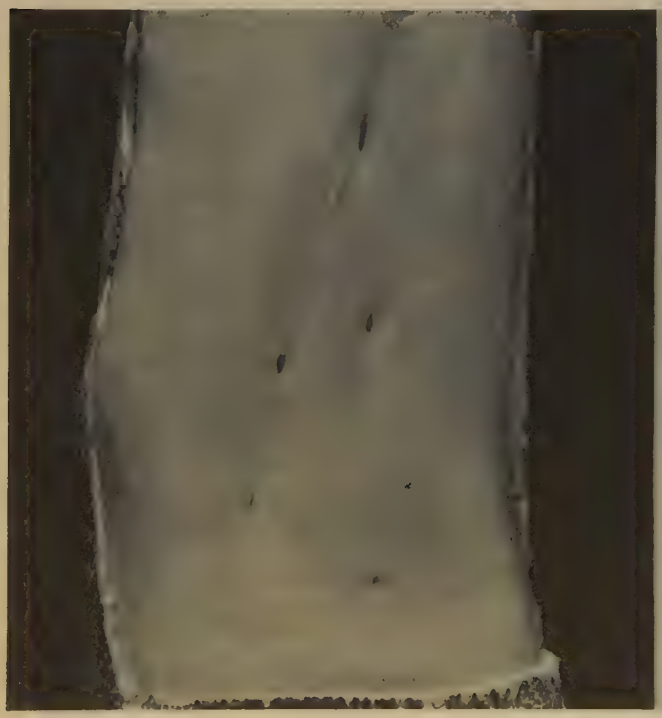

FIGURE 1. Pits in the wood of a 3-year-old trunk of Tien Chieh mandarin seedling inoculated with scions from Wohlford Lisbon lemon. Pegs coinciding with these pits were present on the bark removed from the trunk.

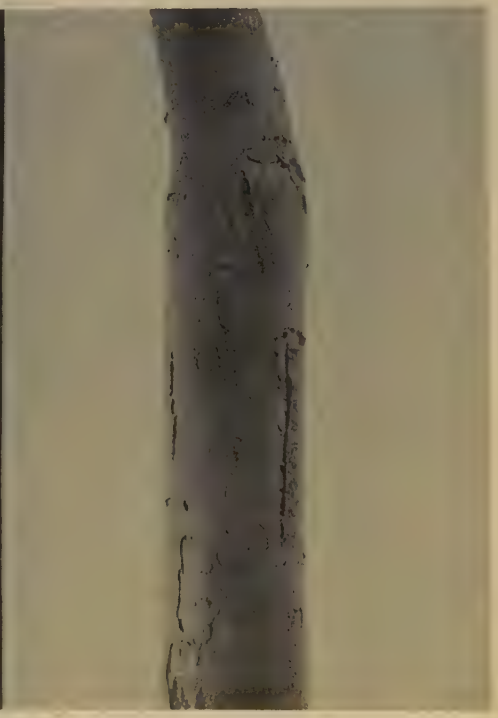

FIGURE 2. Bark cracking in a 3-year-old trunk of Palestine sweet lime rootstock with old-line Eureka lemon top.

An interesting development occurred in Palestine sweet lime stocks grafted with scions from old-line Cascade and C.E.S. Eureka and Wheeler Messina lemon trees. All the test trees developed shallow elongated cracks, resembling growth cracks, 1/4 to 1 inch or more in width, in the outer bark of the trunks (Fig. 2). Removal of a thin section of bark from the cracked area failed to reveal any evidence of xyloporotic wood pitting or other wood or bark abnormalities. Palestine sweet lime stocks with nucellar-line Eureka scions or other lemon scions were not similarly affected.

Table 1. Reaction of cachexia- and xyloporosis-susceptible indicator plants grafted with scions of the indicated lemon selections.

Variety and selections tested[a]	Number of trees tested	Cachexia- and xyloporosis-susceptible plants used		Number of indicator plants showing cachexia or xyloporosis symptoms
		Variety	Number grafted[b]	
EUREKA LEMONS				
Stow Cascade	7	Orlando tangelo	27	0
		Sunshine tangelo	2	0
		Thornton tangelo	9	0
		Wekiwa tangelo	33	0
		Clementine mandarin	19	0
		Tien Chieh mandarin	14	0
		Willowleaf mandarin	14	0
		Palestine sweet lime	12	0[c]
Stow Cascade (seedling-line)	1	Sunshine tangelo	4	0
		Tien Chieh mandarin	5	0
		Palestine sweet lime	12	0
Irvine Cascade	1	Orlando tangelo	6	0
Walden Cascade (seedling-line)	1	Orlando tangelo	6	0
Hughes	2	Orlando tangelo	19	0
		Sunshine tangelo	11	0
		Wekiwa tangelo	9	0
		Clementine mandarin	10	0
		Tien Chieh mandarin	24	0
		Willowleaf mandarin	6	0
Hughes (nucellar-line)	2	Orlando tangelo	12	0
		Sunshine tangelo	8	0
		Thornton tangelo	1	0
		Wekiwa tangelo	7	0
		Clementine mandarin	12	0
		Tien Chieh mandarin	31	0
		Willowleaf mandarin	7	0
		Palestine sweet lime	2	0
Sawyer	2	Orlando tangelo	13	0
		Sunshine tangelo	13	0
		Thornton tangelo	7	0
		Wekiwa tangelo	7	0
		Tien Chieh mandarin	1	0
Sawyer (seedling-line)	2	Sunshine tangelo	5	0
		Thornton tangelo	3	0
		Tien Chieh mandarin	2	0
C.E.S.	2	Orlando tangelo	7	0
		Sunshine tangelo	6	0
		Wekiwa tangelo	2	0
		Clementine mandarin	6	0
		Tien Chieh mandarin	8	0
		Willowleaf mandarin	5	0
		Palestine sweet lime	6	0[c]
Meek	1	Orlando tangelo	5	0
Price	1	Tien Chieh mandarin	2	0
Wheatley	5	Sunshine tangelo	14	0
		Clementine mandarin	2	0
		Tien Chieh mandarin	10	0
Rubidoux	5	Orlando tangelo	7	0
		Sunshine tangelo	3	0
		Thornton tangelo	1	0
		Wekiwa tangelo	7	0
		Clementine mandarin	5	0
		Tien Chieh mandarin	10	0
Allen	1	Orlando tangelo	6	0
		Tien Chieh mandarin	3	0

Table 1 (Continued)

Variety and selections tested[a]	Number of trees tested	Cachexia- and xyloporosis-susceptible plants used		Number of indicator plants showing cachexia or xyloporosis symptoms
		Variety	Number grafted[b]	
Sespe	5	Sunshine tangelo	11	0
		Tien Chieh mandarin	3	0
U.S.D.A.	5	Clementine mandarin	5	0
		Tien Chieh mandarin	3	0
Chase	1	Wekiwa tangelo	3	0
Ross	3	Orlando tangelo	12	0
		Tien Chieh mandarin	9	0
U.C.L.A. #4 (seedling-line)	1	Orlando tangelo	6	0
U.C.L.A. #24 (seedling-line)	1	Orlando tangelo	6	0
U.C.L.A. #4 (seedling-line)[d]	1	Orlando tangelo	3	0
Frost (seedling-line)	1	Orlando tangelo	6	0
		Sunshine tangelo	6	0
Frost (seedling-line)[e]	3	Sunshine tangelo	6	0
		Thornton tangelo	4	0
		Clementine mandarin	3	0
		Tien Chieh mandarin	9	0
		Willowleaf mandarin	3	0
Eureka (miscellaneous old-line selections)	8	Orlando tangelo	23	0
		Sunshine tangelo	5	0
		Clementine mandarin	4	0
		Willowleaf mandarin	4	0
LISBON LEMONS				
Ledig	1	Orlando tangelo	6	0
Walker	1	Orlando tangelo	6	0
Monroe	1	Palestine sweet lime	6	0
Prior 14-18	1	Orlando tangelo	2	0
Prior 22-1	1	Orlando tangelo	6	0
Frost (nucellar-line)	1	Orlando tangelo	6	0
Bevens	1	Sunshine tangelo	5	0
		Tien Chieh mandarin	3	0
Fritz	1	Sunshine tangelo	5	0
		Tien Chieh mandarin	3	3
Wohlford	1	Sunshine tangelo	5	0
		Tien Chieh mandarin	3	3
Limoneira	3	Orlando tangelo	8	0
		Thornton tangelo	10	0
		Wekiwa tangelo	4	0
		Clementine mandarin	5	0
		Tien Chieh mandarin	10	0
		Palestine sweet lime	5	0
MISCELLANEOUS				
Wheeler Messina	1	Orlando tangelo	9	0
		Sunshine tangelo	2	0
		Thornton tangelo	5	0
		Wekiwa tangelo	4	0
		Clementine mandarin	5	0
		Willowleaf mandarin	4	0
		Palestine sweet lime	11	0[c]
Brewer seedling	1	Orlando tangelo	6	0

[a]Selections not specifically designated as seedling-line are old-line selections.
[b]Indicator plants were graft-inoculated in 1954 and 1955. Minimum duration of tests is 3 years.
[c]Longitudinal cracking developed in bark of the trunks of Palestine sweet lime plants. See text for explanation.
[d]Inoculated in 1949 with buds from old-line Eureka tree.

DISCUSSION

It should be pointed out that the indexing tests reported are for 3 to 4 years only and are not completed. It must be recognized that in the different citrus areas virus strains may exist, that different viruses, virus mixtures, or vectors occur, and that symptoms are affected by variations in environment. It may well be that sufficient time has not elapsed for cachexia and xyloporosis symptoms to develop in all of the various test plants under the conditions of the experiment. However, under these same conditions, cachexia has developed within 3 years on Sunshine tangelo and Tien Chieh mandarin trees grafted with scions from known cachexia-infected trees (1).

Present evidence indicates that the virus or viruses of cachexia and xyloporosis are not widespread in commercial varieties of lemons in California. While some of the selections tested are not commercially important at present most of the trees tested represent a cross section of bud-source trees of varieties that comprise much of the total commercial lemon acreage of California. These results with lemons do not coincide with the findings on other species of citrus in Florida (6) and Texas (8, 9) where cachexia was found in a high percentage of the sweet orange (C. sinensis) and grapefruit (C. paradisi) trees grown in those areas.

A growth-retarding virus is known to be present in all of the old-line Eureka lemon trees used in these tests. Furthermore, each of the old-line Eureka lemon source trees tested already was showing symptoms of shell bark or was known to have been propagated from a shell-bark-susceptible parent tree. The failure of cachexia or xyloporosis symptoms to appear in sensitive indicator plants when inoculated with tissue from these old-line Eureka trees indicates that cachexia and xyloporosis probably are not causal factors in the poor vigor of old-line Eureka lemons or in the development of lemon shell bark.

The cause of the bark-cracking in Palestine sweet lime stocks with the old-line C.E.S. and Cascade Eureka and Wheeler Messina tops is not known. The absence of typical xyloporosis symptoms in these test trees and failure of these same scions to induce symptoms of cachexia in susceptible indicator plants suggest that the bark cracks resulted from some other cause. Since only the exocortis virus has been found to be consistently present in the source trees which induced the bark cracking and since inoculations from nucellar-line Eureka scions, known to be generally free of exocortis, did not produce this symptom, it seems probable that the exocortis virus may have been responsible for the bark-cracking in Palestine sweet lime.

Symptoms of the cachexia-xyloporosis type developed on Tien Chieh mandarin seedlings that were inoculated by means of tissue-grafts from two lemon selections (Fritz and Wohlford) which show no symptoms of these diseases. This type of symptom did not develop on seedlings of this indicator variety that were inoculated from other lemon trees. While these two particular lemon selections are not commercially very important they have been propagated to a limited extent in some areas. It seems probable, therefore, that some trees of other selections and varieties, including commercial varieties not thoroughly tested, may be symptomless carriers of the viruses. This re-emphasizes the recommendation that only virus-indexed sources of lemon trees should be used in commercial propagation.

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A QUICK TEST FOR KWANZAN-SYSTEMIC VIRUS[1]

Harold E. Williams and H. K. Wagnon[2]

Programs for the development of fruit tree nursery stock certified to be free of serious virus diseases depend upon methods of indexing for the detection of latent viruses, and generally only a minimum number of indicator plants are used because of the time and expense involved. Rapid and more economical tests for the various virus diseases would permit a wider range of host plants to be used, thus giving greater assurance that certified stock is free of a larger number of specified diseases. The use of Prunus serrulata, varieties Shiro-fugen and Kwanzan, as index hosts for latent ring spot viruses in stone fruit varieties has been reported by Milbrath and Zeller (2, 3) and by Milbrath (1). Both Shiro-fugen and Kwanzan, as index hosts, generally have been accepted as tools in finding clones of stone fruit varieties free of certain latent viruses. The Kwanzan index test has required that individual trees be inoculated during late summer and the results read several months later after the trees have gone through winter dormancy.

It was thought that the cost involved in propagation and maintenance of Kwanzan test trees in experimental plots could be reduced if a quick index method could be devised using greenhouse facilities. Evidence that such a technique might be developed was obtained during the course of some ringspot cross-protection experiments conducted at Oregon State College (5). Mazzard seedlings of Prunus avium were topworked with Kwanzan buds during the summer of 1955 and, at the same time, were bud-inoculated from trees known to be infected with the virus or viruses capable of inducing the epinastic leaf symptom in Kwanzan. Early the next spring, before growth had started, the seedlings were dug and transferred to cans so that they could be forced into growth under greenhouse conditions. The seedlings were examined at digging and many of the Kwanzan buds had died. Therefore, a Kwanzan scion with two to three buds was grafted during February 1956 onto each of those seedlings where the original Kwanzan buds had failed. All trees were placed immediately in a greenhouse and forced into growth. Union between the Kwanzan scion and the mazzard seedling took place within a very short time, and as the Kwanzan leaves unfolded they displayed the epinastic leaf symptom.

A modification of this technique by the senior author, working in California, was to top-work dormant mazzard seedlings by bench whip-grafting in late winter (February 1957) with Kwanzan scions and, at the same time, to inoculate the mazzard seedlings with chip grafts from known ring spot sources (Fig. 1, A). The ring spot source trees had not been tested previously for the virus causing the systemic reaction (epinasty) in Kwanzan. The inoculation chip grafts were placed on the mazzard seedlings in such a manner that each one was vertically aligned with a bud on the Kwanzan scion. A Kwanzan bud not directly above an inoculation chip graft may develop normal foliage in a test of this type.

Three dormant mazzard seedlings which had received Kwanzan whip grafts were chip graft-inoculated on February 26, 1957 from each one of seven ring spot source trees. These plants then were stored in plastic bags at a temperature of approximately 33° F for a period of 1 week, after which they were planted in 1-gallon cans and placed out-of-doors, where they remained dormant. After 2 weeks, on March 13, 1957, they were moved into the greenhouse. Readings were made on April 9, 1957, at which time the epinastic leaf symptom was evident in all the trees in one set of the experiment (Fig. 1, B and C). The time interval from inoculation and whip grafting to good symptom expression was 42 days. Although only one ring spot source of the seven tested resulted in positive symptoms on the test trees, it was thought that other sets might become positive following a dormant period. However, when the trees were examined a year later, in March 1958, no additional positive cases had appeared.

The procedure described in this report can be employed in the winter time, when field conditions are not climatically suitable, provided greenhouse facilities are available. It could supplement a field indexing program. If greenhouse facilities are not available, the test trees could be chip graft-inoculated during dormant season, planted early in the spring in an outdoor plot or in containers set out-of-doors, and readings then made during a period of 1 to 2 months

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[2]Respectively, Assistant Plant Pathologist, and Associate Plant Pathologist, of the Bureau of Plant Pathology, California Department of Agriculture, Sacramento.

FIGURE 1. Kwanzan-grafted mazzard seedlings. A -- Schematic drawing showing method of grafting and inoculation. B -- Seedling at left showing symptoms of systemic virus on Kwanzan leaves. Compare with healthy check plant on right. C -- Close-up view of symptomatic leaves of the Kwanzan scion. Note the extreme twisting of leaves.

after inoculation. However, in order to obtain readings this soon, it is essential that the inoculations be made before the test trees break dormancy. Otherwise symptoms may not appear until the following growing season.

Normal-appearing clones of Kwanzan, as well as Shiro-fugen, have been shown to be carriers of a little-cherry-type virus (4). Since the virus produces a serious disease in fruiting cherries, neither Kwanzan nor Shiro-fugen should be grafted or budded into trees of the fruiting varieties. For the same reason, fruiting varieties should not be used for topworking Kwanzan or Shiro-fugen trees.

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CYPREX: A SUPERIOR CONTROL FOR SHOTHOLE DISEASE OF CHOKECHERRY[1]

R. E. Inman and J. L. Weihing [2]

INTRODUCTION

The shothole disease of chokecherry seedlings has posed a serious threat to nursery stock in past years. The chokecherry was used extensively in shelterbelt plantings of 1936 to 1942, and on the basis of its performance is considered to be an adaptable, desirable plant for providing a low, dense component of windbreaks. In Nebraska the chokecherry is increasing in importance as a source of wildlife food, and serves a minor role in ornamental plantings. Under conditions favorable to disease development, outbreaks of the shothole disease, caused by the ascomycetous fungus, Coccomyces lutescens, have severely reduced the marketability of nursery seedlings, in some cases resulting in total loss of first-year plantings.

Infected leaves first show chlorotic lesions which later become necrotic and fall out, leaving the leaf with a typical shothole appearance (Figure 1). Under more severe infections the typical shothole aspect does not appear; rather, the coalescence of closely spaced lesions produces a general blighting of the leaf and subsequent defoliation.

FIGURE 1. Typical shothole symptoms on first-year chokecherry seedlings.

In the spring and summer of 1958 a spray program was designed to test the effectiveness of several fungicides in the control of this disease.

METHODS

First-year chokecherry seedlings, seeded in the fall of 1957 at the Plumfield Nurseries in Fremont, Nebraska, were sprayed with eight fungicides. The fungicides and dosages used are given in Table 1. Four replications were drawn from a randomized block for each treatment. The first spraying was conducted May 11, 1958, 2 weeks after the seedlings had emerged. The last spraying was conducted September 3. A total of nine sprayings with the prescribed fungicides was made, following at approximately 2-week intervals between May 11 and September 3. The first three sprayings were applied with a Hudson knapsack sprayer at 20 to 25 pounds per square inch (p. s. i.). The remaining sprays were applied with a Century pressure sprayer at 200 to 250 p. s. i. in order to provide complete coverage of the increasing foliar surface.

At times throughout the duration of the spraying, readings were taken upon the various aspects of the disease. Five readings each were taken upon incidence and severity, four on defoliation, and one on plant vigor. Scales were prepared for each disease aspect to be considered, and are as follows:

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[2]Graduate assistant and Extension Plant Pathologist, respectively.

Table 1. Fungicides screened for control of chokecherry shothole disease.

Fungicide	Manufacturer	Active Ingredient	Form	Dose/ 100 Gal.
Cyprex	American Cyanimide Co.	Dodecylguanidine acetate 70%	Wet. Pow.	1 lb.
Manzate	DuPont	Maneb (Manganese ethylene bix-dithio carbamate) 70%	" "	2 lbs.
Actispray	Upjohn	Actidione (B-(2-(3.5-dimethyl) -2-hydroxyethyl)-glutarimide) 7.7%	Tablet	2 p.p.m.
Parzate	DuPont	Zineb (Zinc ethylenebis-dithiocarbamate) 65%	Wet. Pow.	2 lbs.
Orthocide 50	California Spray Chemical Corp.	Captan (N-trichloromethyl-mercapto-4-cyclohexene-1,2-dicarboximide) 50%	" "	4 lbs.
Dyrene	Chemagro Corp.	2,4-Dichloro-6-(O-chloro-aniline)-triazine 50%	" "	2 lbs.
Puratized Agricultural Spray	Gallowhur Chemical Corp.	Phenyl mercury triethanol-ammonium lactate 7.5%	Liquid	1 pt.
Bordeaux mixture	Locally prepared	$CuSO_4$ and Lime	Powders	5 lbs.; 7 lbs.

Incidence
0 -- No infection, or trace.
1 -- Only few older leaves spotted.
2 -- All older leaves spotted.
3 -- All but newest leaves spotted.
4 -- All leaves spotted.

Severity
0 -- No spotting, or trace.
1 -- Light spotting.
2 -- Moderate spotting, no blighting.
3 -- Heavy spotting, some blighting.
4 -- Extensive blighting.

Defoliation
0 -- No defoliation.
1 -- Light defoliation.
2 -- Moderate defoliation.
3 -- Heavy defoliation, lower stems bare.
4 -- Complete defoliation.

Vigor
1 -- Above 18 inches in height.
2 -- 12 to 18 inches in height.
3 -- 6 to 12 inches in height.
4 -- Below 6 inches in height.

Readings were taken in each replicate plot, and the readings of the four replicates for each treatment were averaged in order to arrive at a single index rating for each fungicide.

RESULTS

Readings for the treatments and the control are presented in Table 2. Concerning "incidence," "severity," and "defoliation," for which several readings were taken, only the first and last readings are presented. The single reading for "vigor" is also presented. In addition to the disease ratings, the rank of each treatment denoting the order of its effectiveness in comparison with that of the other treatments is also given.

Table 2. Index ratings and comparative rankings of spray treatments[a].

Treatment	Incidence		Severity		Defoliation		Vigor	Overall rating[b]
	June 30	Sept. 8	June 30	Sept. 8	June 30	Sept. 8	Sept. 8	Sept. 8
Cyprex	1.00 1	2.40 1	0.93 1	1.08 1	0.25 1	0.00 1	1.25 1	4.73
Zineb	2.43 2	3.58 2	2.25 3	3.35 2	0.68 2	2.25 2	2.15 2	11.33
Actidione	2.68 4	3.85 4	2.18 2	3.35 4	0.83 4	3.10 5	2.58 3	12.88
Maneb	2.83 6	4.00 5	2.60 5	3.25 2	1.33 5	2.75 3	3.08 5	13.08
Puratized	2.43 2	3.83 3	2.50 4	3.65 5	0.75 3	2.85 4	2.75 4	13.08
Dyrene	2.75 5	4.00 5	2.83 6	4.00 8	1.75 6	4.00 8	3.65 7	15.65
Captan	3.00 7	4.00 5	3.08 7	3.65 5	2.58 7	3.75 6	3.50 6	14.90
Bordeaux	3.40 8	4.00 5	3.33 8	3.75 7	3.08 8	3.85 7	3.68 8	15.28
Check	3.40 8	4.00 5	3.48 9	4.00 8	3.83 9	4.00 8	4.00 9	16.00

[a]Lowest index indicates best disease control.
[b]Overall disease rating (last column) equals sum of September 8 readings.

Cyprex was by far the most effective of the fungicides tested. Throughout the spraying period Cyprex-treated seedlings showed a minimum of disease in all aspects concerned. By mid-September they averaged nearly 18 inches in height and had lost no leaves as a result of the disease, whereas the checks were less than 6 inches in height and completely defoliated (See Figure 2).

FIGURE 2. The tall plants with heavy foliage are Cyprex-treated chokecherries. The check plants in the foreground are defoliated and small.

Zineb (Parzate), maneb (Manzate), Puratized Agricultural Spray, and Actispray (Actidione) may be placed in a group giving moderate control, while Dyrene, captan (Orthocide) and Bordeaux mixture may be classified as giving little or no control. Control plants were a total loss as far as commercial value is concerned.

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THE USE OF MCINTOSH APPLE SEEDLINGS IN THE BIOASSAY OF CANDIDATE FUNGICIDES FOR CONTROL OF APPLE SCAB[1]

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Summary

McIntosh apple seedlings growing in 4-inch pots are useful for testing new fungicides in the greenhouse for the control of apple scab. The advantages over budded apple trees growing in 12- to 14-inch pots are that a greater number of plants can be grown and cared for and they can be easily moved from the benches in the greenhouse to the inoculation chamber and back, thus permitting a greater number of tests per unit of space. Seeds for planting can be removed from McIntosh apples which have been in cold storage, after 2 months. The seedlings are started in sand or Vermiculite, potted, and are ready for use after forming three to four true leaves. A diffuse-type infection develops on the leaves following inoculation with the conidia of Venturia inaequalis. Estimations of the infected areas of the leaves permit evaluation of the effectiveness of the test fungicide. Three plants with a total of 9 to 12 susceptible leaves are used for each test fungicide. Additional tests may be made as new leaves are formed, or the plants can be cut back and inoculated as new leaves develop. Results from numerous tests show a close correlation between the greenhouse apple seedling tests and field tests for fungicidal value, type of action, dosage rates, and phytotoxicity.

INTRODUCTION

Apple scab, caused by Venturia inaequalis, is reported to be the most serious disease of apples in the United States. McCallan (6) considered it the third most important disease of all agricultural crops in this country. In Nova Scotia, Canada it is estimated that $442,000 per annum is spent for fungicides and their application for the control of this disease (1).

Hundreds of new "candidate" fungicides are developed annually, and it would be impractical to attempt to evaluate all of them in the apple orchard for control of scab. Therefore, a simple time- and space-saving method was sought whereby a large number of compounds, found to be fungitoxic in slide germination tests, could be accurately evaluated in the greenhouse with respect to control of apple scab. Greenhouse tests had been worked out with tomatoes and other herbaceous suscepts with appropriate fungi (5, 7, 8, 13), but these tests were of little value in evaluating fungicides for control of V. inaequalis. Hamilton and his associates (2, 3, 10) worked out an ingenious method using 2- to 3-year-old budded apple trees in large pots. This was an adaptation of a method used earlier by Keitt and Jones (4). The procedure, although effective, requires considerable greenhouse space. Tests using McIntosh apple seedlings were developed at the New Hampshire Agricultural Experiment Station (9, 11, 12) for the bioassay of fungicides. The use of apple seedlings proved advantageous and the method has been adopted as a standard procedure at the New Hampshire Station and by several commercial chemical companies in their fungicide development programs. It seems worthwhile to describe it in detail for publication.

MATERIALS AND METHODS

McIntosh apples are harvested and stored for at least 2 months at 32° F. The seeds are removed by cutting the apples almost in half, and twisting the two halves apart so as not to damage the seeds. The seeds are planted in Vermiculite or sterile sand in flats in the greenhouse. They can be planted immediately or they can be placed in tightly capped bottles and held in the freezing compartment of a refrigerator or directly beneath the freezing unit for use as needed. The seedlings are transplanted into 2 1/2-inch rose pots, using pasteurized

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[2]Plant Pathologist and Mycologist, respectively, New Hampshire Agricultural Experiment Station.

soil. When the seedlings are 5 to 6 inches tall they are usually repotted in standard 4-inch clay pots, and are ready for use as soon as they develop new leaves. If the plants are to be used only once and then discarded, they will not require repotting.

Plants of uniform size, preferably 6 to 8 inches tall, are selected for the tests. It is important for each plant to be vigorous and growing rapidly. Only the three or four youngest leaves are susceptible to scab. A dormant plant can not be used for test purposes. Plants can be maintained in an actively growing state by applications of a nutrient solution to the soil and by foliar applications of magnesium sulfate as needed. Supplemental lighting is used if required during the winter months. Powdery mildew, aphids, and mites must be kept under control. Plants that are heavily infested with mites are highly resistant to apple scab infection under greenhouse conditions.

Fungicide applications are made with a DeVilbiss atomizer attached to an air compressor. The atomizer can be held stationary, and each plant moved about in the spray until both surfaces of the susceptible leaves are thoroughly covered, or the plants can be placed on a compound turntable and sprayed with a portable atomizer, first spraying the lower then the upper surfaces of the leaves. Six plants can be sprayed with as little as 100 ml of the dilute test fungicide. This is a distinct advantage when availability of an experimental compound is limited.

Tenacity of formulated fungicides can be tested by subjecting the fungicide-sprayed plants to "artificial rain." The spray deposit is allowed to dry thoroughly on the leaves prior to inoculation. The "rain" is applied to the plants at a constant temperature and pressure. A TAT No. 6 solid cone nozzle mounted above the compound turntable is used to deliver the "rain," and a rain gauge is used to measure the amount of water applied.

Conidia of V. inaequalis, washed from infected plants previously used as controls, are used for inoculum. The spore suspension, adjusted to approximately 150 spores per microscopic field (X 100), is sprayed onto the dry foliage.

The inoculated plants are held in the moist chamber for 24 hours at 70° F and 90 to 100 percent relative humidity. Temperature-controlled water is circulated from a tank at the bottom to the top of the chamber where it runs down nylon curtains lining the four inside walls of the chamber, thus maintaining a constant temperature and humidity without washing off either the inoculum or the spray deposit.

After the initial incubation period in the chamber, the plants are moved to the greenhouse benches. Apple scab develops on the leaves within 10 to 14 days under greenhouse conditions.

The method described is used to evaluate chemicals as protectant fungicides. Their eradicant action or post-inoculation control (sometimes called "kickback action") can be determined by modifying the procedure. In these tests unsprayed plants are inoculated, incubated for the desired length of time in the moist chamber, then removed and sprayed with the test fungicides. This procedure enables one to measure the property of the fungicide to eradicate or inhibit infection with greater accuracy than can be determined under field conditions.

Scab control can be evaluated in several ways. The pathogen develops in a diffuse pattern on the leaves of the seedlings. The infected leaf areas are estimated and recorded in percentage for each leaf. These percentages are added and the total divided by the number of susceptible leaves, to obtain a mean percentage for all of the susceptible leaves. Three plants are used per fungicide treatment, with a total of 9 to 12 susceptible leaves. The data can be analyzed statistically. Control plants will have at least 50 to 60 percent of the leaf areas covered with scab when the tests are conducted properly.

DISCUSSION AND CONCLUSIONS

The seedlings can be used in two or three consecutive tests before cutting them back if it is assumed that the chemicals are not absorbed to give systemic protection to the new growth. This practice may involve some risk, and it would be safer to cut the seedlings back after each test. Plants are cut back to one or two buds per plant. If more than one shoot develops from the buds only the most vigorous shoot is permitted to grow. Plants are ready for use again in about 4 to 6 weeks after they are cut back. Some plants have been reused for 2 years or more before being discarded. Abnormal plants are removed periodically and are replaced with young, actively growing ones.

Variation in susceptibility to apple scab has not been a problem in the use of McIntosh seedlings. Only one highly resistant seedling has been found among the thousands that have been inoculated.

The tests, as outlined, have proved valuable for determining fungitoxicity, phytotoxicity,

tenacity, and effective dosage rates for new fungicides at an early stage in fungicide test programs. The use of apple seedlings in the test program has saved commercial companies and research workers both time and money.

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CONTROL OF POTATO SEED-PIECE DECAY[1]

D. F. Crossan[2]

Summary

Bacterial and/or Fusarium seed-piece decay may be expected to reduce stands of potatoes when planting is followed by environmental conditions resulting in cool, wet soils. The use of captan alone as a dust or dip treatment will greatly reduce seed-piece decay under such conditions. The use of captan in combination with streptomycin will also benefit seed pieces that must be stored for several weeks or more, resulting in more uniform stands of plants. In well-drained soils, or where planting will not occur until soil temperatures are warm and soil moisture not excessive, freshly cut pieces without chemical treatment have been found to give stands equal to treated seed pieces.

INTRODUCTION

Approximately 10,000 acres are devoted to potato production in Delaware. Most of the acreage is planted in early spring; as a consequence, growers experience reduction in stand due, primarily, to soft rot (Erwinia carotovora) if prolonged cool, wet periods follow planting.

During April 1956, 1957, and 1958 several chemical treatments and methods of handling cut seed pieces of the Cobbler variety of potato were evaluated for control of seed-piece decay at the University of Delaware Research Farm, Newark, Delaware. The individual experiments and results are presented chronologically by year of experiment.

EXPERIMENTATION AND RESULTS

1956

In 1956, five treatments of either 1-minute wet dips or a dust were compared in a split-plot design with irrigation as the main plot effect replicated three times and chemical treatment as sub-plots replicated six times. One inch of water was applied to the irrigated plots 15 days after planting. The seed pieces were cut, treated, allowed to dry, atomized with a suspension of Erwinia carotovora, and planted the same day at the rate of 35 seed pieces per replicate. Treatments and results of stand counts are presented in Table 1.

Table 1. Influence of chemical treatment and irrigation on potato seed-piece decay.

Treatment	Concentration	Average number of plants emerged May 31, 1956[a]	
		Irrigated	Unirrigated
Untreated	-----	19.7	30.0
Captan 50W (dip)	2-100	31.7	33.0
Streptomycin (dip)	100 ppm	31.0	33.3
Captan + streptomycin (dip)	2-100+100 ppm	31.7	33.0
Captan + streptomycin (dust)	2% + 0.1%	31.3	33.0

L.S.D. (.05). Within treatments, 3.7; irr. vs. unirr., 5.8.

[a]35 pieces planted in each of six replicates on April 16, 1956. Irrigated treatments received 1 inch of water on May 1, 1956.

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The application of 1 inch of water resulted in conditions favorable to soft rot development. All chemical treatments gave equal and significant control of Erwinia seed-piece decay under wet conditions; under relatively dry conditions the use of chemical treatment was barely non-significant and the magnitude of difference between untreated and treated rows was considerably less than in the irrigated plots.

1957

In 1957, the method of handling the cut seed pieces was investigated in conjunction with chemical treatment: (a) treated, stored in open storage for 26 days, and planted; (b) stored for 26 days, treated, and planted; and, (c) potatoes cut in pieces, treated, and planted the same day. The treatments consisted of wet dips of 1-minute duration or a dust. The planting site was at the same location as in 1956. A split-plot design with time of treatment as the main plot effect replicated three times, and chemicals as sub-plots replicated three times was used. Treatments and results of stand counts are presented in Table 2.

Table 2. Influence of chemical treatment and method of handling on potato seed-piece decay.

Treatment	Concentration	Average number of plants emerged May 20, 1957[a]		
		A[b]	B[c]	C[d]
Untreated	------	26.3	25.3	30.0
Captan (talc;50%) (dip)	2-100	28.6	29.3	30.0
Talc (dust)	1/2 pound - 100 pounds seed	26.3	26.3	29.0
Captan ($CaCO_3$;50%W) (dip)	2-100	28.6	26.6	29.3
Streptomycin (dip)	100 ppm	27.0	27.3	29.7
Captan + streptomycin (dip)	2-100 + 100 ppm	28.0	26.6	29.0
Captan (dust)	7.5%	26.7	25.0	29.0
Streptomycin (dust)	0.1%	28.7	28.3	28.0
Captan + streptomycin (dust)	7.5% + 0.1%	29.3	28.0	30.0

L.S.D. (.05). Within chemicals, 1.76; between time of treatment, 2.65.

[a]30 seed pieces planted in each of three replicates on April 16, 1957.
[b]Seed treated, stored 26 days, and planted.
[c]Seed stored 26 days, treated, and planted.
[d]Potatoes cut, treated, and planted on same day.

The data show that the method of handling seed pieces prior to planting had a direct bearing on number of plants that emerged. Since no attempt was made to inoculate the seed pieces, it can only be assumed that bacterial soft rot caused the reductions in stand. Treating seed pieces after storage or treating seed pieces before storage were less desirable methods of handling than cutting, and planting the same day. The use of chemical treatment of seed pieces was only beneficial in the case of stored potato pieces. Freshly cut, untreated seed pieces gave perfect stands in this test.

1958

The 1958 test was located near to, but on a different site from, the 1956 and 1957 tests. The purpose of this test was to compare captan and streptomycin dusts for control of Fusarium seed-piece decay (Fusarium solani) as well as bacterial seed-piece decay. Two separate, concurrent experiments were performed using randomized block designs of four replicates each. The potatoes were cut into pieces, air-dried, treated, and placed in open rows. A suspension of either Erwinia carotovora or Fusarium solani was sprayed over the seed pieces and the rows closed. One day after planting, all replicates were irrigated with 2 inches of water. The treatments and results of stand counts are presented in Table 3.

Captan alone, or in combination with streptomycin, controlled both Fusarium and bacterial seed-piece decay. Streptomycin alone controlled bacterial decay but failed to control Fusarium seed-piece decay. This is in agreement with work at other stations (1, 2).

Table 3. Control of Erwinia and Fusarium seed-piece decay by captan and/or streptomycin dust.

Treatment	Amount	Average number of plants emerged June 2, 1958[a]	
		Fusarium	Erwinia
Untreated	-------	28.3	25.0
Captan (8% dust)	4 ounces/50 pounds seed	31.8	31.0
Streptomycin (0.12% dust)	4 ounces/50 pounds seed	27.8	31.5
Captan + streptomycin (8%+0.12%)	4 ounces/50 pounds seed	32.0	31.8
L.S.D. (.05)		1.82	3.35

[a]33 seed pieces per treatment per replicate planted on April 23, 1958.

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DELAWARE AGRICULTURAL EXPERIMENT STATION, NEWARK

VARIETAL RESPONSE TO SEED PIECE DECAY[1]

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Abstract

Although potato varieties differ in susceptibility to seed piece damage (as indicated by percent stand), the differences were not consistent in 2 years of tests. Plants from rotted seed pieces apparently yield less than those from sound seed.

INTRODUCTION

The varied and uncertain performance of seed treatment chemicals (3) suggests that a great many factors are involved in seed piece decay. Some of this variability undoubtedly is due to inherent differences between varieties. Several studies of the resistance of different potato varieties to bacterial rots (1, 2, 5, 6) indicate that clones differ widely in this respect. However, these were the results of laboratory and greenhouse tests and did not reflect the influence of the pathogens on plant performance in the field. In order to obtain information on this point experiments were made in 1956 and 1958 with 12 commonly grown potato varieties.

MATERIALS AND METHODS

The 12 potato clones included in this study are those named in Tables 1 and 2. These varieties represent a rather wide range of maturity classes and some have been found to differ in resistance to bacterial soft rot (4).

The tubers of each variety were divided into three lots: one lot was cut, inoculated and stored for a number of days before planting; another was cut and inoculated in a similar way 1 day before planting. A non-inoculated check, also cut the day before planting, was included.

Table 1. Percent emergence as affected by inoculation of cut seed at different time intervals before planting.

Variety	Emergence as percent of total seed planted: Seed not inoculated		Emergence as percent of emergence from non-inoculated seed pieces: Seed pieces inoculated; indicated number of days before planting			
			1 day	1 day	14 days	4 days
	1956	1958	1956	1958	1956	1958
La Soda	90	99	100 NS[a]	94 NS	93 NS	51 **
Red Warba	97	99	90 NS	91 NS	69 **	0 **
Waseca	91	95	90 NS	93 **	55 **	0 **
Pontiac	95	100	86 NS	87 **	99 NS	0 **
Triumph	95	97	96 NS	68 **	71 **	0 **
Kennebec	95	84	97 NS	47 **	88 NS	0 **
Cherokee	98	97	90 NS	59 **	61 **	0 **
Chisago	90	99	83 *	96 NS	45 **	0 **
Red Kote	97	95	77 **	85 NS	52 **	0 **
Cobbler	90	99	78 *	96 **	29 **	0 **
Chippewa	90	94	79 *	12 **	56 **	0 **
Sebago	71	69	47 **	50 **	33 **	0 **

[a]Student-Neuman-Kuehls Multiple Range Test.
NS = non-significant.
* = significant at 5% level.
** = significant at 1% level.

[1]Paper No. 4089, Scientific Journal Series, Minnesota Agricultural Experiment Station.
[2]Research Fellow, Teaching Assistant, and Professor, respectively, Department of Plant Pathology and Botany, University of Minnesota.

Table 2. Yields per plant, expressed as percent of non-inoculated check, of 12 potato varieties grown from seed inoculated with decay organisms.

Variety	Inoculated 1 day before planting		Inoculated 14 days before planting
	1956	1958	1956
La Soda	100	82	78
Red Warba	105	89	85
Waseca	94	80	88
Pontiac	96	100	92
Triumph	104	87	88
Kennebec	100	91	100
Cherokee	100	110	100
Chisago	95	80	91
Red Kote	111	89	125
Cobbler	100	85	95
Chippewa	90	11	109
Sebago	115	83	92

In 1956, cultures of Erwinia atroseptica (Van Hall) Jennison, Erwinia carotovora (Jones) Holland, and four isolates of tuber-rotting Fusarium spp. were grown on potato-dextrose agar. Spore or bacterial suspensions were prepared from these cultures by washing them with distilled water. Inoculum for the 1958 tests consisted of spore suspensions of four isolates of tuber-rotting Fusarium spp. prepared as in 1956 and a bacterial suspension prepared by mixing soft-rotted potato tubers with water. In both years the inoculum was prepared just before it was used and kept in an ice-water bath to retard spore germination.

Approximately 100 freshly-cut seed pieces of each variety were placed in wire-bottomed flats 18 x 24 inches and the inoculum was applied with a sprayer powered by a portable air compressor. The time and manner of application were standardized to make the inoculum dosage as uniform as possible. The inoculated seed pieces were allowed to dry a few minutes and then put into cloth sacks.

In the 1956 tests the inoculated seed was stored 14 days prior to planting in a metal box with a loose cover at about 75° F. This procedure simulated extreme conditions under which cut seed might be kept on a farm when conditions prevented prompt planting. In the 1958 tests the cloth sacks were placed inside polyethylene bags, the tops of which were loosely knotted, and then stored at 75° F. The polyethylene bags prevented inoculum from soaking through from one cloth sack to another and provided approximately uniform storage conditions for each variety. There were only 4 days of storage before planting in the 1958 test.

Each series of three treatments, early and late inoculation and check, were planted in six replicates in a randomized block split plot in the field. Each plot comprised 15 seed pieces planted 15 inches apart.

RESULTS AND DISCUSSION

Plant Stand

Varietal differences in percent stand or emergence were not the same in 1956 and 1958. Table 1 shows that when planted 1 day after inoculation, four varieties, La Soda, Red Warba, Waseca, and Pontiac, apparently were resistant in both years. Although there were statistically significant differences between the inoculated and check plants of Waseca and Pontiac in 1958, these differences were relatively small. Triumph, Kennebec and Cherokee were resistant in 1956 and susceptible in 1958. The reverse was true of Chisago, Red Kote and Cobbler, although the differences between the two years were not great. Chippewa and Sebago were susceptible in both years.

It is unsafe to conclude from these results that there are real differences in resistance to seed piece decay between any of these varieties. Although four varieties were consistently resistant and two consistently susceptible, when the 12 varieties are ranked each year according to percent emergence, the correlation coefficient for position in the ranks is $r = .070$, which, of course, is non-significant, and indicates that the apparent consistencies may have been due to chance.

Differences in the data from 1956 and 1958 could have resulted from many factors, including inoculum and weather conditions, each of which were different in the two years. In 1956 the seed pieces were inoculated with pure cultures of Fusarium spp. and soft rot bacteria; in 1958 the pure culture of Fusarium was used, but the source of bacteria was a slurry of material from rotted potatoes. The most striking difference in the weather was the rainfall during the 6 days after planting, which was 2.42 inches in 1958 and only 0.11 inches in 1956. This would tend to favor seed piece decay in the field in 1958, as compared with 1956.

The condition of the seed may also vary from year to year and affect susceptibility to decay. This is indicated by the behavior of the variety Kennebec, which produced a 95 percent stand in 1956 from the non-inoculated seed, but only 84 percent in 1958. In 1958 inoculation reduced the emergence of this variety more than it did in 1956. Emergence of Sebago from non-inoculated seed was poor in both years, and it also appeared most susceptible in both years.

Under similar conditions the 12 varieties behaved more consistently than they did in two different years. This may be seen by comparing the results of holding inoculated seed 1 and 14 days before planting in 1956 (Table 1). The relative ranks of the varieties in these tests are somewhat in agreement, the correlation coefficient being .827. Both inoculum and growing conditions were the same in this comparison.

By storing part of the inoculated seed for 4 days instead of for 14 days before planting in 1958 it was hoped to have a test a little less severe than the one of 1956. However, field conditions, probably the greater rainfall right after planting, resulted in almost complete destruction of the seed in 1958. The seed were moderately to badly decayed before planting, as they were after 14 days in 1956.

Yields per Plant

Yields per plant apparently are reduced if the seed piece is decayed. This is shown by Table 2, which presents average yields per plant as percentages of those from the corresponding non-inoculated checks. Where the stand was imperfect, lack of competition would be expected to result in higher yields per plant, other things being equal. In 1956 this seemed true only if stands were considerably reduced, like that of Sebago, planted 1 day after inoculation, or Red Kote in the same test (see Table 1). Planted 14 days after inoculation in the same year, the yields per plant were all less than those from non-inoculated seed, except for Red Kote and Chippewa. This was true even though the stands of some varieties were considerably reduced, and indicates that the surviving plants were injured. In 1958 the effect when planted 1 day after inoculation was more marked than the similar test in 1956. Stands from inoculated seed were lower than in the comparable test in 1956, and yields were also lower.

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EFFECT OF SEED TREATMENT WITH STREPTOMYCIN ON GOLDEN ACRE CABBAGE SEEDLINGS

Huey I. Borders[1]

Summary

Golden Acre cabbage seed treated with streptomycin at strengths as low as 25 ppm produced seedlings that were purple or purple-yellow in color and died after reaching a height of about 1/2 inch. Seeds treated with 15, 10 and 5 ppm were slightly discolored at the start but produced normal plants. Seeds treated with hot water, Arasan, Delsan, Dynactol and the antibiotics Terramycin and Aureomycin produced normal plants.

During the fall and winter of 1955 the efficacy of certain seed treatments for control of plant pathogens borne on the surface of cabbage seeds was tested in the greenhouse. Observations were made also on the possible phytotoxic effects of these materials on seeds and seedlings from the treated seeds. In this and all the later experiments tests were laid out in randomized, replicated blocks. Golden Acre cabbage seed was used throughout the experiments.

The first experiment, conducted September 27, 1955, included the standard hot water treatment and treatments with thiram (Arasan) Delsan (thiram 60 percent, Dieldrin 15 percent), Aureomycin, and mercuric chloride at the dosages and times shown in Table 1. Seeds were also treated with Agri-mycin, a mixture of streptomycin and Terramycin, applied for 30 minutes at dosages of 1:1000, 1:2500, 1:5000, and 1:10,000 (Table 2).

In this experiment the percentage of emergence from seeds treated with Agri-mycin at concentrations of 1:1000 and 1:2500 was much lower than that of untreated seeds and slightly lower at 1:5000 and 1:10,000. After emergence all of the seedlings from seeds treated with Agri-mycin were purple and died by the time they had reached a height of 1/2 inch without ever having developed a normal green color. The other treatments caused varying amounts of reduction in germination as compared with untreated seeds (Table 2) but the seedlings grew as vigorously as those from untreated seeds.

After this effect of Agri-mycin was observed, the experiments were repeated on November 8, 1955. The same materials and dosages were used in one set of treatments, but in a second set the application of Agri-mycin was reduced from 30 minutes to 15 minutes in order to determine whether the shortened treatment caused a different reaction. Since the seeds treated for 15 minutes also produced purple seedlings that died by the time they had reached a height of 1/2 to 3/4 inch, the 30-minute treatment was used in subsequent experiments (Table 2).

As Agri-mycin is a mixture of streptomycin and Terramycin, the next experiments (January 1956) included these antibiotics as separate materials in order to determine whether either material alone would cause purpling and death of the seedlings. Trials were made also with Dynactol and reduced dosages of Aureomycin (Table 2). Treatments with hot-water and Arasan were included also in these and all later experiments. Treatments with Terramycin and streptomycin showed that streptomycin, not Terramycin, caused the effects observed in the preceding experiments.

In tests in 1957, cabbage seeds soaked 30 minutes in solutions containing 400, 200, 100, 50, or 25 ppm of streptomycin and planted in greenhouse beds germinated in slightly greater numbers than the untreated checks, but the seedlings that emerged ranged in color from a solid deep purple from seeds treated with the higher concentrations to a mixed purple-yellow color from seeds treated with a concentration of 25 ppm. All these seedlings reached a height of about 1/2 inch and then died (Table 2).

Seeds treated with streptomycin concentrations of only 15, 10, and 5 ppm produced seedlings that ranged in color from light purple-yellow to yellowish-green. All of these seedlings eventually became green and grew normally. Seed treatments with materials other than strep-

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Table 1. Golden Acre cabbage seed treatments, 1955-1957.

Material	Concentration	Manner of application	Subsequent handling of seed
Hot water		Soaked 25 minutes at 50° C then dipped in cold water	Dried
Arasan	4 ounces per 100 pounds of seed	Dust treatment	
Delsan	Seed sprinkled with a slurry of 3 ounces in 1 pint of water	Seeds sprinkled with the slurry and thoroughly mixed.	Dried
Agri-mycin[a]	1:1000, 1:2500, 1:5000, 1:10,000, 100 ppm, 75 ppm, 50 ppm	Soaked 30 minutes	Dried
Agri-mycin[a]	1:1000, 1:2500, 1:5000, 1:10,000	Soaked 15 minutes	Dried
Streptomycin[b]	400 ppm, 200 ppm, 100 ppm, 50 ppm, 25 ppm, 15 ppm, 10 ppm, 5 ppm	Soaked 30 minutes	Dried
Terramycin[c]	400 ppm, 200 ppm, 100 ppm, 50 ppm	Soaked 30 minutes	Dried
Aureomycin[d]	1:1000, 1:2500, 1:5000, 1:10,000, and 100 ppm, 75 ppm, 50 ppm	Soaked 30 minutes	Dried
Dynactol[e]	1000 ppm, 200 ppm, 100 ppm	Soaked 30 minutes	Dried
Mercuric chloride	1:1000	Soaked 10 minutes then rinsed 5 minutes in running water	Dried

[a]Agri-mycin = A mixture of streptomycin (15 percent) and Terramycin (1.5 percent) furnished by Charles Pfizer & Co., Inc.
[b]Phytomycin = A 20 percent streptomycin nitrate solution furnished by Olin Mathieson Co.
[c]Terramycin = Soluble Terramycin, (235 mcg/mg), furnished by Charles Pfizer & Co., Inc.
[d]Aureomycin = Aureomycin hydrochloride, Technical, furnished by American Cyanamid Company.
[e]Dynactol = Monoxychlorosene, furnished by Guardian Chem. Corp., Long Island, City, N. Y.

tomycin caused no discoloration of seedlings or other evident physiological effects and seedling emergence and subsequent growth were normal in comparison with those of seedlings from the untreated seeds (Table 2).

A field plot experiment (Table 2), which included the same set of treatments used in the greenhouse experiment of February 11, 1957, yielded the same results as were obtained in the greenhouse.

It is hoped that the description of this syndrome will be of value to workers interested in the physiological effects of streptomycin on plants and other biological systems.

Table 2. Percent emergence, seedling color, and growth of Golden Acre cabbage from treated seeds, 1955-1957.

Treatment	Percent emergence							Seedling color[b]	Growth pattern[c]
	Number of seeds and date of treatment								
	Greenhouse					Field			
	400 9/27/55	800 11/8/55	800 11/8/55	800 1/6/56	800 1/9/56	400 2/11/57	400 2/11/57		
Hot water	27	20	21	54	80	57	50	G	N
Arasan	26	43	53	70	68	71	50	G	N
Delsan	31	43	47			76	72	G	N
Agri-mycin 1:1000	15	41[a]	28					P	D
Agri-mycin 1:2500	20	37[a]	34					P	D
Agri-mycin 1:5000	36	47[a]	29					P	D
Agri-mycin 1:10,000	35	28[a]	38					P	D
Agri-mycin 100 ppm				70				P	D
Agri-mycin 75 ppm				70				P	D
Agri-mycin 50 ppm				65				P	D
Streptomycin 400 ppm					68	74	70	P	D
Streptomycin 200 ppm					68	72	65	P	D
Streptomycin 100 ppm					67	72	75	P	D
Streptomycin 50 ppm					69	74	71	P	D
Streptomycin 25 ppm						78	66	PY	D
Streptomycin 15 ppm						75	68	YG	N
Streptomycin 10 ppm						50	70	YG	N
Streptomycin 5 ppm						70	67	YG	N
Terramycin 400 ppm					72			G	N
Terramycin 200 ppm					66			G	N
Terramycin 100 ppm					70			G	N
Terramycin 50 ppm					67			G	N
Aureomycin 1:1000	32	42	31					G	N
Aureomycin 1:2500	39	49	39					G	N
Aureomycin 1:5000	40	46	42					G	N
Aureomycin 1:10,000	36	46	38					G	N
Aureomycin 100 ppm				62				G	N
Aureomycin 75 ppm				73				G	N
Aureomycin 50 ppm				82				G	N
Dynactol 1000 ppm				73				G	N
Dynactol 200 ppm				69				G	N
Dynactol 100 ppm				77				G	N
Mercuric Chloride 1:1000	31	50	48	71	67	76	66	G	N
Check, No Treatment	43	35	41	69	71	65	63	G	N

[a]Agri-mycin applied as a 15-minute soak instead of 30 minute soak.
[b]G = Green; P = Purple; PY = Purplish Yellow; YG = Yellowish Green.
[c]N = Normal growth of seedling.
D = Seedlings died at height of 1/2 to 3/4 inch without producing a green color.

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THE EFFICACY OF CERTAIN SYSTEMIC COMPOUNDS IN THE CONTROL OF ASPARAGUS RUST[1]

Harry H. Murakishi

Summary

Of seven experimental compounds tested, Acti-dione-S (semicarbazone analog of cyclohexamide) and D-113 (1,2 dichloro 1-methyl sulfonyl ethylene) were the most promising against asparagus rust. Plants sprayed with these two compounds had greener foliage and better needle retention than plants sprayed with other compounds or than the unsprayed control. In further tests at two locations, two applications of Acti-dione-S were nearly equal to six applications of zineb in controlling rust. The dosage of Acti-dione-S that gave good control without apparent phytotoxicity was .1 quart at 100 ppm per plant. Slight to moderate injury occurred at twice the concentration (200 ppm) or when the volume of spray at 100 ppm was increased from .1 quart to .25 quart per plant. However, all plants initially injured by the excess dosages of spray recovered within 1 month. The results with zineb are in agreement with earlier findings.

Protective spraying with zineb has been demonstrated to be effective in controlling asparagus rust (Puccinia asparagi DC.). Van der Vliet (5) in Holland reported significant increases in yield over a 2-year period when zineb was applied on a 10- to 14-day schedule. In northern Illinois, Linn and Lubani (4) found that eight applications of zineb at about 8-day intervals were superior to six applications.

Studies on chemical control of other foliar diseases have shown that certain systemically active compounds impart a longer period of protection, thereby allowing a substantial reduction in number of applications. Hamilton, Szkolnik and Sondheimer (2) reported that derivatives of Acti-dione afforded at least 3 weeks of systemic protection to cherry trees from Coccomyces leaf spot. Hacker and Vaughn (1) stated that the semicarbazone analog of cyclohexamide, Acti-dione-S, induced long-term resistance to wheat plants from attacks of stem rust, increased the yield, and was only slightly phytotoxic. The present paper reports results of a field screening test to determine the efficacy of Acti-dione-S and certain other systemic compounds on the control of asparagus rust in 1957, and tests comparing the two most promising compounds with zineb as the standard treatment in 1958.

FIELD SCREENING OF SYSTEMIC COMPOUNDS, 1957

Procedure

The compounds (Table 1) were applied at relatively high concentrations since the primary aim was to obtain maximum control with only two applications. Preliminary greenhouse trials had indicated that the concentrations tested in the field were only slightly phytotoxic when applied to young seedlings of the Viking and Mary Washington varieties. In the greenhouse, injury was expressed as a leaf-tip dieback from which all seedlings sprayed with any of the compounds tested appeared to recover normally within 2 to 3 weeks.

The spray plots in 1957 were located at the Southwest Michigan Experimental farm at Sodus. Plants of the variety Viking planted in 1955 were spaced 18 inches in the row and 4 feet between rows. Each treatment row, replicated four times with two guard rows in between,

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contained 20 plants. The field was harvested on May 2, 7 and 8, after which the plants were allowed to develop fern growth. In spite of the dense foliage and the presence of dew, uredia did not develop until the first week of August. The two applications with the compounds were made using a 3-gallon hand-pressure sprayer, the first on August 14 and the second on August 23. Approximately .5 gallon of spray mixture was needed to cover both sides of a treatment row to the point of run-off. Since rust appeared late in the season, evaluation was confined to the most recent stalk to emerge after spraying began following a method developed by Kahn et al. (3) in which 0 represented no infection and 10, very heavy infection. In addition, compounds were rated as to their effect on the foliage.

Table 1. Evaluation of experimental compounds in controlling asparagus rust (uredial and telial stages) after two applications on the variety Viking at Sodus in 1957.

Experimental compounds[a]	Ppm of active ingredient applied	Mean infection indices of stems[b]	General appearance of foliage[c]
C.P. 8525	1250	2.28*	Slightly yellowish-green
C.P. 8621	1250	1.81**	Reddening of leaf-tips and stalks
C.P. 9425	1250	2.22**	Do
C.P. 10512	1250	1.59**	Do
C.P. 10616	1250	2.66	Slightly yellowish-green
Acti-dione-S[d]	200; 100	.48**	Good green color
D-113	1250	1.44**	Good green color
Control	----	3.71	Moderate to severe yellowing
L.S.D. .05		1.09	
L.S.D. .01		1.48	

[a]C.P. 8525 = ethionine, C.P. 8621 = acrolein phenylhydrazone, C.P. 9425 = 4-ethylthio-2-hydroxyl butyramide, C.P. 10512 = 5-chloro-2(2-diethylamino-ethylthio) benzothiazole, C.P. 10616 = 2(2-diethylaminoethylthio) benzothiazole (Monsanto Chemical Company), D-113 = 1,2,dichloro 1-methyl sulfanyl ethylene (Chemagro Corporation), Acti-dione-S = semicarbazone analog of cyclohexamide (The Upjohn Company). All compounds except D-113 were designated by manufacturers as having either systemic or post-infection properties against other rusts. Two cc of Triton B-1956 was added to each gallon of spray.

[b]Mean infection indices determined on September 13 were based on 25 plants per row replicated 4 times.

[c]As determined on September 13.

[d]First application of Acti-dione-S was at 200 ppm, the second at 100 ppm.

* Significant at 5% level.

** Significant at 1% level.

Results

As shown in Table 1, Acti-dione-S was the most effective of all seven compounds tested in controlling rust. Although slight to moderate phytotoxicity was noticed after the first application at 200 ppm, the plants soon recovered. The second application at 100 ppm caused no injury. At the end of the test, Acti-dione-S and D-113-sprayed plants had greener foliage and had retained their needles better than had plants from other treatments. These two compounds were therefore selected for further testing. In decreasing order of effectiveness, Acti-dione-S, D-113, C.P. 10512, C.P. 8621 and C.P. 9425-sprayed rows had significantly less rust than the control at the .01 level. C.P. 8525 had significantly less rust than the unsprayed at the .05 level. C.P. 10616 was ineffective.

COMPARISON OF ACTI-DIONE-S AND D-113 WITH ZINEB, 1958

Procedure

Acti-dione-S and D-113, judged to be the most effective of the compounds tested in 1957, were selected for further testing at Sodus and at East Lansing in comparison with zineb. At

Table 2. Effect of Parzate, D-113 and Acti-dione-S in controlling asparagus rust (uredial and telial stages) on the variety Viking at Sodus in 1958.

Fungicide[a]	Rate of application	Number of applications	Mean infection indices of stem[b]
Parzate	2 pounds/100 gallons	6	.73
D-113	1250 ppm	2	2.91
Acti-dione-S	100 ppm	2	1.73
Control	-----		4.40
L.S.D. .05			1.01
L.S.D. .01			1.34

[a]Parzate (E. I. duPont Company) = 65% zineb. D-113 = 1,2,dichloro 1-methyl sulfanyl ethylene. Acti-dione-S = semicarbazone analog of cyclohexamide. Two cc of Triton B-1956 was added to each gallon of spray.
[b]Mean infection indices determined on October 10 were based on 10 plants from a 20-plant-row replicated three times.

Table 3. Effect of Dithane Z-78, D-113 and Acti-dione-S in controlling asparagus rust (uredial and telial stages) on the varieties Viking and Mary Washington at East Lansing in 1958.

Fungicide[a]	Rate of application	Number of applications	Mean infection indices of stem[b]
Variety Viking:			
Dithane Z-78	2 pounds/100 gallons	6	1.93
D-113	1250 ppm	2	2.91
Acti-dione-S[c]	100; 10 ppm	2	1.68
Control	----		4.88
L.S.D. .05			.54
L.S.D. .01			.71
Variety Mary Washington:			
Acti-dione-S[c]	100; 10 ppm	2	2.35
Control	----		5.84
L.S.D. .05			.61
L.S.D. .01			.81

[a]Dithane Z-78 (Rohm and Haas Company) = 65% zineb -- six weekly applications beginning July 25. D-113 was applied on July 25 and August 13. Acti-dione-S was applied on July 25 at 100 ppm and on August 25 at 10 ppm. Two cc of Triton B-1956 was added to each gallon of spray.
[b]Mean infection indices based on a 20 plant row replicated three times.
[c]First application of Acti-dione-S was at 100 ppm; the second at 10 ppm.

Sodus the field plot layout was similar to that previously described. In 1958 this plot was harvested during April 23 to May 14. Uredia did not appear until the week of July 28. Acti-dione-S at 100 ppm was applied twice (August 5 and 14). D-113 was also applied on these dates. Zineb (Parzate) applications were made six times on a 7-to 10-day schedule from August 5 to September 15. As in the previous test, a 3-gallon hand-pressure sprayer was used and approximately .5 gallon of spray mixture was needed to cover both sides of a treatment row to the point of run-off. Prior to fungicide applications, all rusted stalks were tagged and eliminated from the subsequent rust evaluations made on October 10. Rust readings were taken on one to four stalks per plant and the average of the readings was recorded.

At East Lansing, the field was not harvested. Uredia were first observed on the week of July 21. In contrast to the test at Sodus, a 15-gallon power sprayer[2] was used instead of the hand sprayer. About 1.5 gallons of spray was used to cover each 25-plant row to the point of run-off. Beginning July 25, zineb (Dithane Z-78) was applied on a 7-to 10-day schedule until a total of six applications had been given, the last on September 4. D-113 was applied on July 25 and August 13. The first application of Acti-dione-S at 100 ppm was on July 25, but the second was postponed until August 25 because moderate phytotoxicity was noted on both the Viking and Mary Washington varieties. Rust evaluations were recorded on September 29.

Results

In the test at Sodus, no injury was caused by the two applications of Acti-dione-S at 100 ppm. Although six applications of zineb (Parzate) gave somewhat better control than Acti-dione-S, the difference was not statistically significant (Table 2). D-113 was the least effective of the treatments but it was still significantly better than the control at the .01 level.

At East Lansing on the variety Viking, Acti-dione-S and zineb (Dithane Z-78) were about equally effective in controlling rust (Table 3). On the variety Mary Washington, only Acti-dione-S was tested but its rust-controlling properties were again demonstrated. The injury caused by the first application of Acti-dione-S at 100 ppm with the power sprayer was probably due to the more than twice the volume of spray (.25 quart per plant) applied at East Lansing than that given at Sodus (.1 quart per plant).

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[2]Maximum capacity of 3 gallons per minute.

THE EFFECT OF AMOUNT OF WATER USED IN APPLICATION ON FUNGICIDAL EFFICIENCY OF A RUST CONTROL CHEMICAL[1]

Bjorn Peturson and F. R. Forsyth[2]

Summary

In 1957 and 1958 two protectant fungicides (nabam + zinc sulphate and zineb 65 percent) were applied in varying amounts of water to control cereal rust in field plot experiments. Although the control of rust was not good, because rust infection had occurred before spraying commenced, the sprayed plots in both years yielded much more and produced grain of higher kernel weight and bushel weight than the non-treated plots. In the 1957 test the plots sprayed at the rate of 5 and 10 gallons per acre gave the best results; those sprayed at the rate of 50 and 100 gallons per acre were not significantly better than the check.

In the 1958 test the plots sprayed at the rate of 50 gallons per acre gave the best yields and produced the best grain, although there was no significant difference between spray treatments.

The results of these tests show that the fungicides used in these tests are at least as effective for rust control when applied in 10 gallons of water as when applied in higher gallonages.

INTRODUCTION

The amount of water required per acre in applying a rust control fungicide could have a decisive bearing on the practicability of its use for cereal rust control on an extensive scale. If cereal rust control by chemical methods were to become a general farm practice in the grain-growing areas of Western Canada large acreages of cereals would be treated. The quantity of water needed is an important cost factor and if large amounts of water were required for the fungicidal applications the use of aeroplanes for this purpose would be impractical. As there appeared to be no reference to this problem in the literature, experiments were carried out at Winnipeg, Manitoba in 1957 and 1958 to determine the amount of water, as a carrier of a good rust fungicide, that would give best rust control.

The present paper gives the results of these tests.

METHODS

The highly rust-susceptible wheat variety Red Bobs was used in both years. It was grown in the field in rod-row plots. Each treated plot consisted of four rod-rows and was separated from other test plots by four-row buffer plots of Red Bobs. There were four randomized blocks in the 1957 test and six in the 1958 test. All of the test plots in each block, except the checks which received no treatment, were sprayed with tank-mix zineb (nabam, 1 1/2 quarts, plus zinc sulphate, 3/4 pound per acre), in the 1957 test, and with zineb 65 percent (2 pounds per acre) in the 1958 test. The fungicide was applied to the plots in the following volumes of water per acre: 5, 10, 25, 50 and 100 gallons. In the 1958 test the plots receiving 5 gallons of spray per acre were sprayed once accidentally with a different spray material and were discarded. Four applications of the fungicides were made in the 1957 test and six in 1958. In both tests about a 1 percent rust infection was present when the first application was made.

The fungicides were applied with a knapsack sprayer of 1-gallon capacity, except for the 5 gallon per acre rate which was applied with an atomizer owing to the difficulty of applying this small quantity with the knapsack sprayer.

Certain plots in experiments adjacent to the test plots were artificially infected with both leaf and stem rust of wheat and the rust that developed in this test came mainly from this source.

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[1]Contribution No. 18 from the Canada Department of Agriculture Research Laboratory, Winnipeg, Manitoba.

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The effectiveness of the different sprays was appraised by determining: 1) the amount of rust present in each treatment, 2) the 1000-kernel weight, and 3) the bushel weight and the yield of each plot.

RESULTS AND DISCUSSION

Rust was not satisfactorily controlled chiefly because too many rust infections had taken place before spraying was started. However, in both years the onset of heavy infection was delayed by the sprays and although the sprayed plots became heavily infected and were severely damaged they yielded much more and produced grain of higher kernel and bushel weight than the non-treated checks. The results of both tests are given in Table 1.

Table 1. Result of applications of tank-mix zineb in 1957 and zineb (65 percent) in 1958 in various amounts of water for rust control.

Amount of water used per acre in applying fungicides (gallons)	Leaf and stem rust (percent)	Average yield of seed per acre (bushels)	Increase in yield of seed over check (percent)	Weight per 1000 kernels (grams)	Increase in kernel weight over check (percent)	Average weight per bushel (pounds)	Increase in bushel weight over check (percent)
1957 Experiment:							
Check	90	7.8	---	10.40	--	42.5	----
5	40	11.6*	49	17.18**	65	49.8**	7.3
10	45	15.0**	92	17.74**	70	50.5**	8.01
25	65	11.1*	42	16.87**	62	48.0*	5.5
50	80	9.9	27	13.09**	26	47.5*	5.0
100	85	8.3	6	11.32	9	44.5	2.0
L.S.D. 5%		3.5		1.64		4.1	
L.S.D. 1%		4.9		2.22		5.6	
1958 Experiment:							
Check	95	5.2	---	9.09	--	35.8	----
10	50	14.5**	179	15.10**	66	45.6**	9.9
25	65	15.0**	188	15.81**	74	43.8**	8.0
50	65	16.4**	216	16.16**	78	46.3**	11.5
100	65	15.4**	196	17.17**	89	45.3**	10.0
L.S.D. 5%		2.7		2.10		3.1	
L.S.D. 1%		3.6		2.83		4.2	

* Significant at 5% level.
**Significant at 1% level.

In the 1957 test best results were obtained in the plots that received the lower gallonages, the plots receiving the fungicide in 10 gallons of water per acre producing the best yields. The plots to which the fungicide was applied in 50 and 100 gallons of water per acre were not significantly better than the check.

In the 1958 test all the treated plots, although severely damaged by rust, yielded very much more than the check. Although there was no statistically significant difference between the various sprays, the one in which the fungicide was applied at the rate of 50 gallons per acre was slightly better than the others.

These tests indicate that the rust control fungicides used are just as effective, if not more effective, when applied in low volumes of water (10 gallons or less per acre) than when applied in high volumes of water (50 to 100 gallons per acre). It is not known why the fungicide gave poorer rust control in the 1957 test at the higher gallonages, but the difference in physical properties of tank-mix zineb prepared in various amounts of water may have been the deciding factor.

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EFFECT OF SEED TREATMENT OF COTTON WITH THIMET, A SYSTEMIC INSECTICIDE, ON SEEDLING DISEASES IN THE FIELD

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Summary

Seed treatment of cotton with Thimet 44D in the absence of a fungicide caused a statistically significant reduction in the stand of seedling plants in four of seven field tests conducted in several cotton-growing areas of California. Stands were improved when seeds were treated with a fungicide (Ceresan 200, Ceresan M, or captan) prior to adding the Thimet coating. However, only in tests conducted in the Imperial, Palo Verde, and Coachella valleys did all of the Thimet-fungicide treatments result in seedling emergence comparable to that obtained from seed treatment with a fungicide alone. In other tests, only at Arvin was the treatment Thimet-captan comparable to that from a fungicide alone.

In 1957 frequent losses of stand of cotton occurred in the San Joaquin Valley, California when seed was treated with the systemic insecticide, Thimet 44D, in addition to some of the common fungicides. In 1958 results of greenhouse studies (2) showed that stand loss due to seed treatment with Thimet and Ceresan 200 occurred in cool (61° F) non-sterilized soil but not in sterilized. These studies also showed that treatment of seed with a combination of captan and Thimet resulted in higher percentages of emergence than treatment with Thimet and Ceresan 200. Similar results have been obtained in tests with a number of different field soils from the San Joaquin Valley.

A seed treatment test was conducted in several areas of California in March and April of 1958 to compare the Thimet-captan seed treatment with other Thimet-fungicide combinations and to test the effect of Thimet on germination of cotton seed in the field. Results of these tests are presented in this paper.

METHODS

Acid delinted Acala 4-42 cotton seed (95 to 97 percent germination) was treated with the fungicides by pouring the liquid or slurry on the inside surface of a glass jar (about twice the volume necessary to contain the seed) followed by agitation of seed and fungicide for 3 minutes. The method of treating seed with Thimet was reported previously (2).

Active ingredients of chemicals and dosages per 100 pounds of cotton seed were:

Captan 75W -- 75 percent N-(trichlormethylthio)-4-cyclohexene-1,2-dicarboximide, 2 ounces.

Ceresan 200 -- 6 percent ethyl mercury 2,3-dihydroxy propyl mercaptide and 1.3 percent ethyl mercury acetate, 1 and 2 ounces.

Ceresan M -- 7.7 percent N-(ethylmercuri)-p-toluenesulfonanilide, 2 ounces.

PCNB -- 75 percent pentachloronitrobenzene, 4 ounces.

Thimet 44D -- 44 percent O, O diethyl S-(ethylthiomethyl) phosphorodithioate formulated on activated charcoal, 8 pounds.

Approximately 200 seeds were planted in each row-plot, 50 feet in length, by hand dropping the seed through planting tubes of commercial planters. The test was designed as a random-

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ized block with six replications. Plant counts were made two or three times after emergence and numbers of plants dead from seedling disease were recorded. The data presented are the percentages of seedlings that emerged from 200 seeds planted. Percentages were transferred to angles for analysis, and multiple range tables of Duncan (1) were utilized for evaluating mean differences.

Tests conducted at the United States Department of Agriculture, Southwest Irrigation Field Station in the Imperial Valley, in the Coachella Valley, and in the Palo Verde Valley yielded similar results; so they were combined and designated the "southern California" data. In the San Joaquin Valley, tests were conducted near Arvin, Coalinga, Visalia, and Murray. The data from two tests conducted at the Citrus Experiment Station at Riverside have not been included as they were similar to those from some of the tests conducted in the San Joaquin Valley.

RESULTS

Since the differences between percentages of emergence and survival were slight, only the emergence data are shown in Figures 1-5.

In all tests, except those conducted at Indio, Blythe, or Brawley (Fig. 1), the percentage of emergence from seeds treated with Thimet alone was significantly lower than from no treatment seeds. This indicated that reduction of stand due to Thimet was not entirely the result of the interaction of Thimet with fungicides. An effect of the systemic chemical on the germinating seedling may also be involved.

When a fungicide was applied prior to treatment of seed with Thimet, a greater percentage of emergence occurred in all tests (Figs. 2-5) except in those conducted in southern California, where differences between all treatments were small (Fig. 1). At Arvin (Fig. 3) the Thimet-captan seed treatment resulted in a high percentage of emergence which was not significantly different from the emergence from seed treated with the fungicides alone but significantly greater than that occurring where Thimet was used in combination with either dosage of Ceresan 200 or Ceresan M. Only the Arvin data amongst all data from the San Joaquin Valley tests (Fig. 2-5) agreed with those from the previously run greenhouse tests (2).

The Thimet-captan treatment induced a lower average emergence than did PCNB-captan in all tests except at Arvin (Figs. 1-5). These differences were significant only at the 5 % level. The consistency of these results suggested that Thimet-captan under these field conditions might be less effective than the PCNB-captan treatment. These data, coupled with the low emergence percentages due to Thimet alone, indicated that the fungitoxicity of Thimet against Rhizoctonia, shown in artificially infested soils (2), would not be useful in the field, and that Thimet should not be used as a seed treatment in the absence of a fungicide.

There are no apparent reasons why the percentage of Thimet-treated seeds that emerged was higher in southern California areas than elsewhere. The average soil temperature at the 2-inch depth (obtained from thermograph records for 2 weeks after planting) did not indicate that temperatures were lower in the San Joaquin Valley during the 1958 season than they were in the Coachella Valley. Nevertheless, seed decay and seedling disease due to Pythium spp. are usually more of an economic problem in the San Joaquin Valley than in the southern California desert valleys.

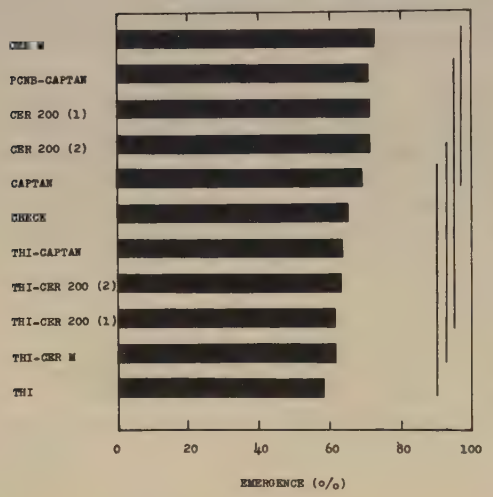
Isolations from diseased seedlings yielded Rhizoctonia solani from the Indio, Brawley, Arvin, Visalia, and Murray tests and Pythium spp. from Indio, Coalinga, Arvin and Murray tests. Plants from seed treated with Thimet did not consistently yield different numbers of either Pythium or Rhizoctonia isolates than those from seed not treated with Thimet.

DISCUSSION

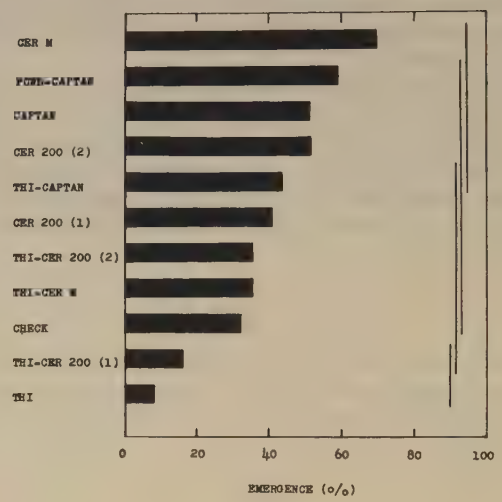
In a previous report (2), it was suggested that the interaction of Thimet with the mercurial fungicides might be responsible for the reduced emergence of Thimet-Ceresan 200 treated seeds but that a possible physiological change in the susceptibility of the seedlings to Pythium should also be considered.

Data from some of the field tests reported here showed an adverse effect of Thimet on germination of cotton seedlings. This suggested that a physiological effect of Thimet might be responsible for stand loss. However, in recent greenhouse studies seed treated with activated charcoal and Ceresan 200 showed a lower percentage of emergence in field soils than seed treated with Ceresan 200 alone. It appeared here that an interaction between the adsorptive charcoal carrier of Thimet and the mercurial fungicide Ceresan 200 might have occurred.

1. SOUTHERN CALIFORNIA



2. VISALIA



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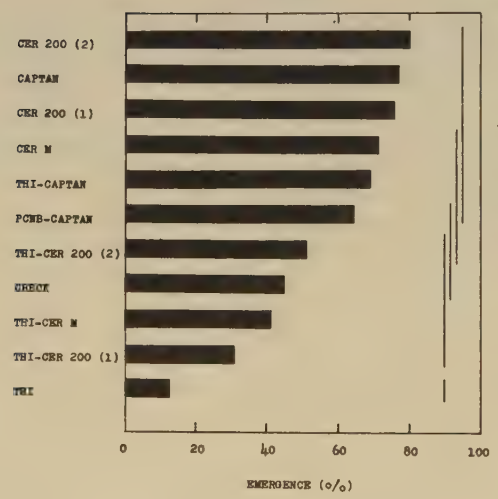
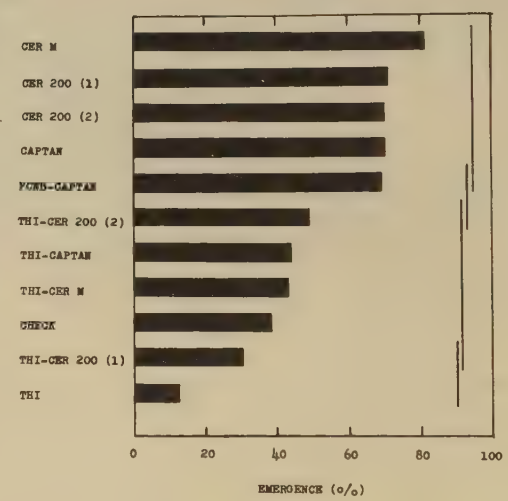
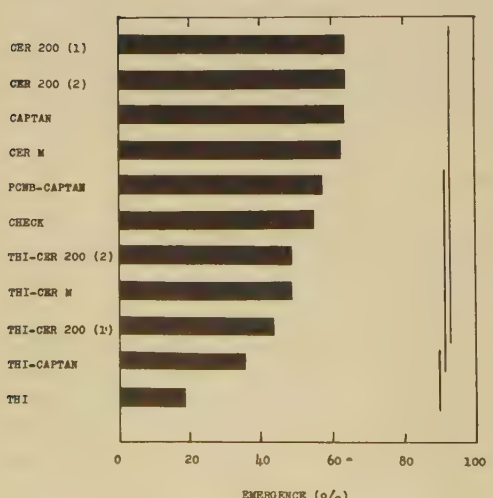


4. COALINGA



5. MURRAY



FIGURES 1-5. Effects of several fungicides alone and in combination with Thimet on emergence of cotton seedlings (percent of 200 seeds planted). Location of tests were as follows: Fig. 1 -- southern California (data from Blythe, Indio, and Brawley combined), Fig. 2 -- Visalia, Fig. 3 -- Arvin, Fig. 4 -- Coalinga, and Fig. 5 -- Murray, California. The number in parentheses following the Ceresan 200 treatment refers to dosage in ounces per 100 pounds of cotton seed. The population classifications (1 percent) of the means are indicated by the lines on right of each graph according to the multiple range test of Duncan (1).

When seed was treated with Thimet 44D (active chemical on charcoal) in addition to Ceresan 200, the reduction in percentage of emergence was even greater. These data, coupled with the reduced stand in the field due to Thimet alone, suggested that Thimet might have induced an adverse physiological effect on cotton seed. Because of its high degree of phytotoxicity, Thimet in liquid formulations was not tested as a seed treatment.

The increased percentage of emergence induced by the Thimet-captan treatment in comparison with the Thimet-mercurial combinations in the test at Arvin and in field soils in the greenhouse might be due to the lesser adsorption of captan by charcoal than of the mercury compounds. The failure of captan in combination with Thimet to increase emergence in some of the other field tests might be a result of the dominance of a physiological effect of Thimet. No quantitative assay of the relative importance of the two factors under different conditions has been made.

Leach et al. (3) reported a reduction in emergence of lima bean seed which had been treated with Lindane (99.95 percent gamma isomer of benzene hexachloride). This reduction, caused by soil-borne seed decay organisms, was either eliminated or greatly reduced by addition of a fungicide such as thiram, bis(dimethylthiocarbamoyl)disulfide, or chloranil, tetrachloro-p-benzoquinone. Their results bear certain similarities to those reported in this paper. The addition of any fungicide increased the stand from Thimet-treated seeds but seldom eliminated the stand depressing effect of Thimet.

The data presented by Leach et al. (3) and by this paper emphasize the great need for thoroughly controlled laboratory, greenhouse, and field testing of systemic and other insecticidal seed treatments in such a way that their effects on the incidence of seed decay or seedling disease can be accurately measured.

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A FIELD EVALUATION OF SIX STICKERS IN COMBINATION WITH PARZATE FOR THE CONTROL OF COFFEE RUST[1]

R. B. Valdez, A. N. Pordesimo, and F. T. Orillo[2]

Summary

A field evaluation of six stickers in combination with Parzate for the control of coffee rust in the Philippines was conducted on 3-year old Coffea arabica trees. The stickers tested were Goodrite p.e.p.s., Triton B-1956, Orthocide sticker 1017, Para rubber latex, Orthocide spreader-sticker, and Shell Tenac. Rubber latex was applied at the rate of 1 1/2 pints while the others were applied at 3 pints per 100 gallons. The sprays were put on at 2-week intervals. Final counts of rusted, defoliated, and healthy leaves were made only on leaf-growth increments starting from the first spray application.

Parzate plus Goodrite p.e.p.s. gave the least rusted leaves with 9.4 percent, followed by Parzate plus Orthocide sticker 1017, Parzate plus Triton B-1956, and Parzate plus Orthocide spreader-sticker with 11.07, 11.53, and 12.75 percent rusted leaves, respectively. Parzate alone gave 18.03 percent and the untreated check 89.81 percent rusted leaves. Analysis of variance showed highly significant differences in all the spray treatments over the untreated check. However, the differences between the treatments with and without stickers were statistically insignificant.

INTRODUCTION

In the Philippines where heavy precipitation occurs and relative humidity is generally high throughout the year, the use of stickers to improve tenacity is indispensable for efficient utilization of foliage fungicides. The spray deposit is, in many cases, washed away by torrential rains. These rigorous conditions often mar the reputations of otherwise proven foliage fungicides in the temperate zone. Properly prepared Bordeaux mixture is still considered the most outstanding protectant fungicide largely because of its excellent sticking qualities and its resistance to tropical weathering. In spite of these inherent qualities, however, published accounts show that even the most favored Bordeaux mixture requires sticker to be more effective.

Mayne, as cited by Wellman (6), was probably the first to show the importance of stickers in the control of coffee rust caused by Hemileia vastatrix Berk. & Br. in India. In two series of Mayne's experiments, the unsprayed trees showed 88 and 87 percent rust infection. The treatment with copper alone gave 73 percent, but a well prepared Bordeaux mixture reduced rust infection to 51 and 49 percent, respectively, in two different trials. Moreover, when stickers were added separately to Bordeaux mixture, rust infection was reduced to 42 percent with casein, 36 percent with resin soda, and 37 percent with linseed oil. In a series of tests for the control of coffee rust in Southern India, Thomas (4) reported that the addition of teepol X spreader at the rate of 1/4 and 1/2 ounce to 2-2-40 Bordeaux mixture also increased leaf retention. The addition of stickers to sprays on coffee has also been pointed out (5) to provide uniform coverage and prevent loss of spray deposits due to rains.

In experiments with stickers combined with Orthocide against Cercospora and Colletotrichum on coffee seedlings in Costa Rica, Havis and his co-workers (1) found that nonsprayed seedlings had 16 percent disease-free leaves; Orthocide alone applied every 20 days, 43 per-

[1]This work was conducted under the Coffee and Cacao Development Project in the College of Agriculture and Central Experiment Station. Project No. 1200.

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cent; Orthocide plus sticker RDA 156, 82 percent; and Orthocide plus Goodrite p.e.p.s., 74 percent. In further trials, they found that when Goodrite p.e.p.s. was added to Fermate it produced over 100 healthy leaves. Fermate alone gave below 70, Goodrite p.e.p.s. plus Perenox gave 62, while Perenox alone gave 52 to 57; in comparison, the nonsprayed gave only 24 healthy leaves.

Apparently stickers play an important role in the control of coffee diseases. At present, there seems to exist a wide variety of commercial stickers. In addition to these, latex from Para rubber, which is locally grown, has been reported (3) as one of the promising stickers selected from 47 spray supplements tested in combination with cupric oxide for the control of potato blight.

Although a number of commercial stickers are available in Philippine markets, their recommended dosage rates are of practically no value in retaining the fungicides. This fact was shown in the bioassay tests conducted by Reddy and Davide (2). Results of their experiments demonstrated that the best rates of these stickers were much above the manufacturers' recommendations. This study was therefore aimed to: 1) carry out under field conditions their formulated laboratory recommendations in combination with Parzate, and 2) compare these combinations with Bordeaux mixture in the control of coffee rust. Bordeaux mixture is effective under tropical conditions, but is inconvenient to prepare. Furthermore, it causes fruit russeting, burning of leaves during cool and wet seasons, increases the rate of desiccation of foliage during drought. This experiment was conducted in the Mas-Arabica Coffee Plantation in Masalukot, Candelaria, Quezon, approximately 75 miles southeast of Manila, from September 1957 to January 1958.

MATERIALS AND METHODS

Three-year old Coffea arabica trees planted at 3 x 3 meters were used in the experiment. The set-up consisted of a randomized block design with nine treatments including the check. Each treatment was replicated five times with six trees per replication.

The stickers tested were, Goodrite p.e.p.s. (50 percent polyethylene polysulfide), Triton B-1956 (Rohm & Haas Co.), Orthocide spreader-sticker (Calif. Spray-Chem. Corp.), Shell Tenac (Shell Chem. Co.), Orthocide sticker 1017 (Calif. Spray-Chem. Corp.), and Para rubber latex (42 percent total solids). Para rubber latex was applied at the rate of 1 1/2 pints per 100 gallons while all others were applied at 3 pints per 100 gallons. Parzate (65 percent zinc ethylene bis[dithiocarbamate]) at the rate of 2 pounds per 100 gallons was used in combination with each of the stickers. A 4-4-50 Bordeaux mixture served as the standard of comparison for the effect of the stickers while Parzate alone and the untreated check served as control. Three-gallon compressed-air sprayers were used throughout the experiment.

Since some of the experimental trees were already rust-infected before the start of the experiment, it was deemed necessary to evaluate the effect of the materials only on the leaf growth increments starting from the first spray application. Markers made of pieces of string were tied on the terminals and laterals of a representative tree in each replication to indicate the leaf growth increments throughout the duration of the experiment. The first spray was applied September 20, 1957 and subsequent sprays were applied at 2-week intervals. A total of six spray applications was made.

Two weeks after the last spray application, the rust reading was made by counting from 130 to 150 leaves on the growth increments of each tree and noting the number of healthy, rusted, and defoliated leaves. Veeder-root hand-tally counters were used to facilitate counting.

RESULTS AND DISCUSSION

A slight difficulty was experienced in the application of the spray with rubber latex, as small particles of coagulated rubber occasionally clogged the spray nozzles. No serious phytotoxic effect was observed with the treatments used except the slight burning exhibited by reddening of narrow areas mostly found along the leaf-margins of trees sprayed with Parzate plus Orthocide spreader-sticker.

The treatment having the fewest rusted leaves was Parzate plus Goodrite p.e.p.s., with 9.4 percent. This was followed by Parzate plus Orthocide sticker 1017, Parzate plus Triton B-1956, and Parzate plus Orthocide spreader sticker with 11.07, 11.53, and 12.75 percent rusted leaves, respectively. The results are summarized in Table 1.

Table 1. Protectant effect of spray stickers in combination with Parzate against coffee rust.

Treatment	Number of leaves counted	Percentage of leaves		
		Healthy	Rusted	Defoliated[a]
Parzate + Goodrite p. e. p. s.	3914	90.60	9.40	3.27
Parzate + Ortho sticker 1017	4354	88.93	11.07	3.86
Parzate + Triton B-1956	4268	88.47	11.53	2.41
Parzate + Ortho spreader-sticker	4049	87.25	12.75	2.40
Parzate + Rubber latex	4487	85.12	14.88	6.26
Parzate + Shell Tenac	4200	84.62	15.38	3.67
Bordeaux mixture	4416	84.04	15.96	5.16
Parzate (alone)	4054	81.97	18.03	5.15
Unsprayed	4425	10.19	89.81	27.39

[a]Defoliation due to rust. Figures included in column "Rusted."

Analysis of variance on the number of rusted, defoliated, and healthy leaves showed highly significant differences in all the treatments over the untreated check at both the 1 and 5 percent levels. However, the differences between the treatments with and without stickers were statistically insignificant. The effect of stickers was not well demonstrated, presumably because the experimental plots were partially shaded by tall coconut trees which protected the coffee plants from direct torrential rains. However, the results obtained indicate that the inherent tenacity of Parzate could still be improved by the addition of the right amount of good stickers, such as any of the materials tested. Parzate in combination with effective adhesives can compare favorably with, if not excel, Bordeaux mixture in the control of coffee rust.

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EFFECT OF SELECTED FUNGICIDE-ASPHALT MIXTURES ON THE GROWTH OF CERATOCYSTIS FIMBRIATA F. PLATANI IN VITRO

Curtis May and John G. Palmer[1]

The effect of eight fungicides mixed with asphalt varnish on the growth of Ceratocystis fimbriata f. platani in vitro was tested as part of a study of possible formulations of antiseptic tree wound paints that could be prepared from readily available fungicides.

Ceratocystis fimbriata f. platani causes a fatal disease of the London planetree. Ceratocystis fimbriata Ell. & Halst. causes black rot of sweetpotato. Moreover a fungus identified as C. fimbriata causes a lethal disease of cacao. However, host specialization is probable. In a preliminary study conducted in a greenhouse at Beltsville, Maryland, no disease developed in twelve young cacao trees inoculated with an isolate of C. fimbriata f. platani obtained from a diseased London planetree.

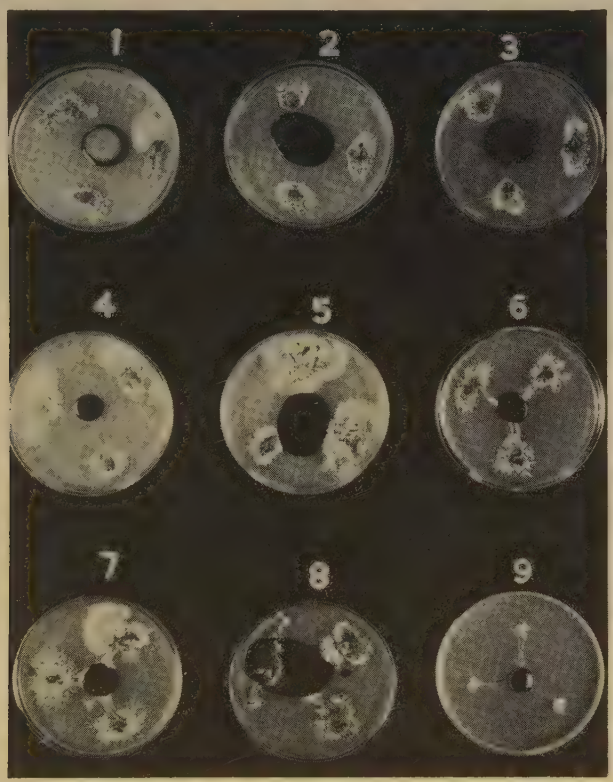

FIGURE 1. Effect of asphalt varnish-fungicide mixtures on growth of Ceratocystis fimbriata f. platani in vitro. Mixtures contain: 1-dichlone, 2-Phaltan, 3-thiram, 4-ferbam, 5-maneb, 6-zineb, 7-Ni-carbamate, 8-no fungicide, 9-phenyl mercury nitrate. The streaking visible in plates 1 to 4 inclusive, and 9, are light reflections from the cuts in the agar.

In the study reported here a culture of C. fimbriata f. platani isolated from London planetree was used[2]. Colonies of the fungus were planted on poured plates of potato-dextrose agar at three places approximately equidistant from the center of the plate and equidistant from

[1] Plant Pathologists, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland.

[2] The authors thank Mr. Lawrence Crone, Research Fellow, Department of Plant Pathology, Rutgers University, for supplying the isolate.

each other. After 4 days a small amount of each paint was placed on the agar in five plates. Three days later streaks were made with a sterile needle from each colony to the paint.

Inhibition of growth of the fungus from the colonies towards the asphalt-fungicide mixture indicated that a compound toxic to the fungus had spread from the mixture into or over the culture medium.

The cultures were observed 1 week after the streaks were made. In plates without paint or with paint to which no fungicide was added, the fungus grew throughout the streak and up to or over the asphalt. Formulations containing 0.5 percent zineb, 0.5 percent maneb and 0.5 percent of the nickel carbamate did not inhibit growth of the fungus. Paints containing 0.5 percent dichlone, 0.5 percent ferbam, 0.5 percent Phaltan, or 0.5 percent thiram prevented growth of the fungus in the streaks (Fig. 1). Phenyl mercury nitrate 0.25 percent also prevented growth of the fungus. Asphalt varnish containing dichlone soon became too stiff to use.

Ceratocystis fimbriata f. platani can be spread from tree to tree in non-antiseptic asphalt tree wound paint. Use of 0.02 percent phenyl mercury nitrate in asphalt varnish is effective in preventing spread of the fungus in the paint. No field trials have been made or are contemplated with tree wound paints containing ferbam, Phaltan, or thiram. However, since these fungicides were effective in the study and are readily available field tests would be of interest.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

FIELD RESISTANCE OF VARIOUS STRAWBERRY VARIETIES[1] AND SELECTIONS TO VERTICILLIUM

E. H. Varney, J. N. Moore and D. H. Scott[2]

Abstract

Fifty strawberry varieties or selections were tested for resistance to Verticillium wilt in a naturally heavily contaminated field. Several varieties and selections were highly resistant. The collapse of a number of plants of resistant varieties such as Blakemore and IVT 667-Juspa suggests that strains of Verticillium differing in pathogenicity exist in eastern United States and that they must be considered in future breeding and evaluation programs.

In recent years Verticillium wilt or "summer dying" of strawberries, caused by Verticillium albo-atrum Reinke & Berth., has been accentuated in New Jersey by the growing popularity of the susceptible variety Jerseybelle. Heavy losses, however, have occurred only in fields previously planted to tomatoes or other highly susceptible crops. Tomatoes and strawberries are the principal crops for many New Jersey growers. Since crop rotations are unlikely to give adequate control (5) and soil fumigation has disadvantages, it appeared that the use of resistant varieties would be the best control measure to follow. This paper reports the results of a preliminary field screening of 50 varieties or selections for sources of resistant commercial varieties and for resistance to be utilized in a breeding program.

METHODS

Field trials were conducted on a farm with a known Verticillium wilt problem. Apples were grown in the test field prior to 1950, tomatoes in 1950-52, wheat in 1953, strawberries in 1954-55, and tomatoes in 1956-57. The experimental plots were laid out in four randomized blocks. Each block included the varieties and selections listed in Table 1, and each plot consisted of 12 plants planted 1 foot apart. Rows were 4 feet apart to permit routine cultivation and fertilization by the cooperating grower. Plants were supplied by the United States Department of Agriculture. Dormant plants were set April 24, 1958.

Individual plants were rated for wilt on August 5 and October 17, 1958. The disease classes recognized were 0, plant apparently healthy; 1, slight stunting, no necrosis; 2, moderate stunting, some necrosis; 3, severe stunting and necrosis; 4, plant dead. A disease index for each plot was obtained by totaling the individual ratings. Mean plot values were converted after analysis to a 0 to 100 scale, in which 0 indicates all plants apparently healthy and 100 all plants dead.

The overall appearance of each plot was also evaluated by using a scale from 0 to 10. Plots with all plants apparently normal in vigor were rated 0 and plots with all plants dead were rated 10. Intermediate grades were used to indicate the percentage of each plot showing normal plant development.

RESULTS

Results of evaluation of individual mother plants on October 17 are shown in Table 1. It is evident that several varieties or selections showed high field resistance. No plants of the Sheldon clone of Fragaria virginiana, Catskill, Vermilion, Md-US 1972, Md-US 2650 and NC 1759 showed severe wilt symptoms or were dead. Only 2.4, 6.3 and 6.8 percent of the Howard 17 (USDA), Surecrop and Aberdeen plants, respectively, and 8.3 percent of the Gem, Howard 17 (Kellogg) and NC 1768 plants were severely affected or dead. In Blakemore, a variety considered resistant, 19.6 percent of the plants were killedor showed severe symptoms.

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[2]Assistant Research Specialist, Research Associate, New Jersey Agricultural Experiment Station; and Principal Horticulturist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, respectively.

Table 1. Field evaluation of various strawberry varieties and selections for susceptibility to Verticillium wilt in New Jersey, October 17, 1958.

Variety or selection	Percent dead or severely wilted	Disease index[a]	Parentage[b]
F. virginiana (Sheldon clone)	0.0	0.0	Wild species
Vermilion	0.0	0.0	Redstar x Pathfinder
Catskill	0.0	1.0	Howard 17 x Marshall
Md-US 1972	0.0	1.6	Blakemore x Md-683
NC 1759	0.0	2.1	US 4127 x Tennessee Beauty
Md-US 2650	0.0	2.6	Md-US 2188 x US 4144
Surecrop	6.3	5.7	Fairland x Md-US 1972
Howard 17 (USDA)	2.4	9.4	Crescent x Howard 1
Aberdeen	6.8	11.5	Chance seedling
NC 1768	8.3	14.6	US 4127 x Tennessee Beauty
Temple	14.6	14.6	Aberdeen x Fairfax
Tennessee Beauty	12.5	15.1	Missionary x Howard 17
Howard 17 (Kellogg)	8.3	16.7	Crescent x Howard 1
Gem	8.3	17.7	
IVT 667-Juspa	18.8	19.3	Jucunda x Sparkle
Md-US 2651	14.6	19.3	Md-US 2188 x US 4144
Md-US 2601	13.0	21.9	US-4152 x Stelemaster
Blakemore	19.6	23.4	Missionary x Howard 17
Md-US 2555	21.7	26.0	Vermilion x Stelemaster
Md-US 2596	23.8	27.1	US 4152 x Stelemaster
Md-US 2408	27.1	27.6	Tennessee Beauty x Temple
Redglow	25.5	29.7	Fairland x Tennessee Shipper
Md-US 2595	29.2	30.2	US-4152 x Stelemaster
Empire	29.2	30.7	Dresden x Sparkle
Fairfax	36.2	37.0	
Fairland	39.6	38.0	Aberdeen x Fairfax
NC 1786	41.7	40.1	US 4127 x Midland
Md-US 2289	43.8	42.7	Fairland x Md-US 1972
Albritton	45.7	44.8	NC 1065 x NC 1053
Md-US 2396	50.0	48.4	Fairland x US 3919
Sparkle	51.1	53.1	Aberdeen x Fairfax
Armore	58.3	56.8	Aroma x Blakemore
Pocahontas	56.3	59.4	Tennessee Shipper x Midland
Md-US 2321	62.5	60.0	Temple x Md-US 1972
Missionary	60.0	60.4	Chance seedling
Md-US 2610	58.3	60.4	US 4152 x Stelemaster
Midland	72.9	65.6	Howard 17 x Redheart
Jerseybelle	70.2	69.3	NJ 953 x NJ 925
Md-US 2445	72.3	70.8	Temple x Md-US 1972
Md-US 2389	74.5	71.3	Dixieland x Temple
Redstar	75.0	71.9	Chesapeake x Fairfax
Md-US 2359	77.1	73.4	Fairland x Midland
Md-US 2606	76.7	76.0	US 4152 x Stelemaster
Md-US 2488	78.7	77.1	Dixieland x Temple
Md-US 2558	80.4	79.7	Stelemaster x Md-US 2162
US 3961	83.3	81.8	Tennessee Shipper x Midland
Dixieland	89.6	90.1	Tennessee Shipper x Midland
Md-US 2675	91.5	90.6	Redglow x Md-US 2210
Earlidawn	95.7	94.3	Tennessee Shipper x Midland
US 4192	100.0	96.9	Fairland x Tennessee Shipper
LSD 19:1		24.9	
LSD 99:1		32.3	

[a]Based on scale in which 0 indicates all plants apparently healthy and 100 all plants dead. (See text.)

[b]Md 683 = Scotland BK 46 x Fairfax
Md 2210 = Md 683 x Midland
Md-US 2162 = Fairland x Md 683
Md-US 2188 = Md-US 1972 x Midland
US 3919 = Tennessee Shipper x Midland
US 4127 = Fairpeake x US 3358
US 4144 = Redstar x Md 430
US 4152 = Tennessee Shipper x Maytime

Verticillium was readily isolated from infected Blakemore plants. In IVT 667-Juspa, a resistant variety from the Netherlands, 18.8 percent of the plants were killed or developed severe wilt symptoms.

Data on the overall appearance of each plot are not included in Table 1, for the values obtained agreed with few exceptions with the disease indexes. Notable exceptions were Howard 17 (USDA), Aberdeen and Temple. These varieties were given relatively low disease indexes, but the overall appearance of the plots at the end of the growing season was poor to fair. It is possible that their poor performance should be attributed to Verticillium. Howard 17 (Kellogg) plots were significantly better in appearance than the Howard 17 (USDA) plots. The most susceptible varieties were also the first to show severe wilt symptoms in June. On November 5 all Earlidawn plants were dead.

DISCUSSION

Resistance and susceptibility of varieties and selections graded into each other with no sharp breaks. As Wilhelm (5) has pointed out, this grading into each other suggests a quantitative type of inheritance. The fact that at least 19.6 percent of the plants of Blakemore, a resistant variety, were dead or severely wilted may indicate a quantitative resistance similar to that of certain cabbage varieties whose resistance to Fusarium yellows is influenced by temperature and nutrition (3, 4). It is also possible that there are in the trade two clones of Blakemore which differ in degree of resistance or that strains of Verticillium more pathogenic to Blakemore than those tested by Wilhelm (5) are present in New Jersey. Wilhelm found that about 17 percent of resistant Blakemore plants were invaded by the pathogen. The breakdown of a number of plants of IVT 667-Juspa, a resistant variety from Holland, also suggests that a variety may differ in its resistance to strains of Verticillium.

The most difficult varieties to evaluate were those that grew poorly but showed few, doubtful, or no diagnostic symptoms of wilt. Howard 17 (USDA), Aberdeen and Temple are good examples of such varieties. Wilhelm (5) included in his susceptible class all Howard 17 plants tested. It will be necessary to determine how extensively plants of these varieties were invaded by the pathogen.

It is interesting to speculate on the possible sources of resistance in the cultivated strawberry whose ancestors are Fragaria chiloensis (L.) Duch. and F. virginiana Duch. Wilhelm (5) found wilt resistance in certain North American clones of F. chiloensis but none in F. virginiana clones or seedlings. Van Adrichem and Orchard (2) also found resistance in progenies of F. chiloensis from Chile. Newton and van Adrichem (1) found no tolerant or resistant seedlings in the F_1 generation of self-fertilized F. virginiana. In view of these reports, the apparent resistance of the Sheldon clone of F. virginiana in a heavily infested soil is of interest. It is suggested that there is inherent resistance in some clones of F. virginiana and that this species may also have contributed resistant genes to the cultivated strawberry.

The extreme sensitivity of the new varieties Dixieland and Earlidawn should be noted, for in some areas Verticillium wilt could be the limiting factor in their acceptance. The highly wilt-resistant Vermilion, Surecrop and Aberdeen are also resistant to one or more strains of the red stele fungus, Phytophthora fragariae Hickman.

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RESISTANCE IN WATERMELON TO COLLETOTRICHUM LAGENARIUM RACES 1, 2, AND 3

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Summary

Eighty-six varieties of watermelon, citron and advanced breeding lines of watermelon were evaluated for resistance to Colletotrichum lagenarium races 1, 2, 3. All varieties resistant to race 1 were found to be resistant to race 3 and susceptible to race 2. The remaining varieties were susceptible to each of the three races. Approximately 350 Citrullus vulgaris Plant Introductions were tested for resistance to race 2. Twenty of these showed slight resistance in one test, but were found to be susceptible in two subsequent tests. An African citron, line W-695, was found to be segregating for resistance to race 2. This was the only line or variety tested which was resistant to C. lagenarium race 2. Inheritance studies showed that resistance to races 1 and 3 is controlled by the same dominant gene. Inheritance studies for resistance to race 2 were inconclusive.

The destructiveness of the anthracnose disease incited by Colletotrichum lagenarium (Pass.) Ell. & Halst. to watermelon, Citrullus vulgaris Schrad., has been markedly reduced during the past few years in North Carolina. This has been primarily associated with the widespread planting of anthracnose resistant varieties such as Charleston Gray, Congo, and Fairfax.

Several investigators have reported the development of anthracnose resistant varieties of watermelon. Layton (11) crossed African watermelon lines 8, 9, and 13 with Iowa Belle, Iowa King and other varieties and reported that resistance to anthracnose was controlled by a single dominant factor pair. From these crosses watermelon breeding lines resistant to anthracnose were developed. In 1938 Firky (7) screened varieties of watermelon for anthracnose resistance in Egypt. Among the varieties included in this report resistance was apparent only in the very early stages of infection. A watermelon, line X-32, developed at the Georgia Experiment Station (4) was reported as continuing to show more resistance to both wilt and anthracnose than other strains. Welch and Melhus (14), in Iowa, screened 85 lines and varieties of watermelon. They found one strain resistant to both wilt (caused by Fusarium oxysporum (Schlecht.) f. niveum (E. F. Sm.) Snyder & Hansen) and anthracnose. While not immune to anthracnose, these lines were alive and produced mature melons 2 weeks after susceptible varieties were dead. Welch and Melhus (15) reported progress in breeding for anthracnose resistance, and Melhus, Reddy, and Emans (13) reported that two early varieties, Early Resistant Queen and Black Kleckley, were moderately and very resistant, respectively, to wilt and anthracnose. In 1956 the anthracnose resistant watermelon variety Black Kleckley was increased for seed supply (12).

The first anthracnose resistant varieties of watermelon to find wide acceptance were Congo, Fairfax, and Charleston Gray, released by Andrus in 1949 (1), 1953 (2), and 1955 (3), respectively. According to Crall (6) these varieties carry the same monofactorial resistance to anthracnose as the resistant breeding lines developed by Layton in Iowa.

Recently there have been reports that anthracnose resistant watermelon varieties were susceptible to this disease. Boelema (5) reported that although Congo was resistant under South Africa conditions, the resistance level was not sufficient during the rainy season or in poorly drained fields.

Goode (8) observed severe anthracnose on leaves, stems and fruits of Congo and Charleston Gray watermelons at four locations in North Carolina in 1954 and 1955. Colletotrichum lagenarium has been isolated from Congo fruits shipped from Florida; these isolates were pathogenic on seedlings of Congo and Charleston Gray (personal communication C. F. Andrus,

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United States Department of Agriculture). From 1955 to 1958 the authors have observed one or more commercial plantings each year of anthracnose resistant varieties in North Carolina severely affected with the anthracnose disease.

Goode and Winstead (10) reported three pathogenicity groups and in 1958 Goode (9) designated these as three physiologic races in C. lagenarium. He reported that plants of Charleston Gray, Congo, and Fairfax were resistant to races 1 and 3 and susceptible to race 2. In addition, plants of Black Kleckley were resistant to race 1 and susceptible to race 2. Plants of the varieties Garrison and New Hampshire Midget were susceptible to all three races. Goode also reported that approximately 20 Plant Introductions and 1 African citron, line W-695, were tolerant or resistant to race 2.

The present investigation was directed towards the screening of available watermelon varieties for resistance to races 1, 2, and 3. In addition, all available Plant Introductions, including those previously screened by Goode, were evaluated for resistance to race 2. Studies were also undertaken to determine the inheritance of resistance to the three races.

MATERIALS AND METHODS

Seedlings of the various varieties and lines were grown in the greenhouse at 26° to 32° C in a soil and sand mixture that had been disinfested with methyl bromide.

The fungus was grown on snap bean agar (400 grams of finely ground cooked green beans), 20 grams agar and enough water to make 1 liter.

The techniques of inoculation were essentially similar to those previously described (9) and plants were usually inoculated in the 2- 3- true-leaf stage. The inoculum concentration was 2500 to 10,000 spores per ml. Isolates used were those previously designated as typical isolates of C. lagenarium races 1, 2, and 3 (9).

RESULTS

Screening Commercial Varieties

Commercial varieties of watermelon were screened for resistance to races 1, 2, and 3. In some cases plants of the same variety from several seed sources were tested. At least 25 plants of each variety were inoculated with each race. The varieties susceptible to races 1, 2, and 3 are listed in Table 1. The varieties and advanced breeding lines resistant to races 1 and 3 and susceptible to race 2 are listed in Table 2. All varieties and lines that were resistant to race 1 were resistant to race 3. No varieties tested were resistant to race 2.

Since resistance to races 1 and 3 was available in commercial varieties, Plant Introduction lines were screened only for resistance to isolates of race 2. Approximately 350 Plant Introductions were obtained from the Southern Regional Plant Introduction Station, Experiment, Georgia. No lines survived inoculation with this race. Anthracnose lesions formed on the foliage and stems of these plants and the plants were all dead within 14 days after inoculation. In each of two subsequent tests, involving approximately 50 plants of each of the 19 lines previously reported by Goode as tolerant or resistant. All Plant Introductions were highly susceptible. The Plant Introductions tested for resistance to race 2 and found susceptible are listed in Table 3. The 19 Plant Introductions previously reported as resistant were: 163572, 164977, 164448, 165451, 169258, 169297, 172789, 175660, 175664, 176488, 176909, 176917, 180427, 183123, 183673, 186490, 211851, 212287, and 222714.

Two types of citron were tested for resistance to race 2. Seed of healthy citron plants growing near Blackville, South Carolina were collected, and approximately 2000 seedlings from this lot were inoculated. Twenty plants remained healthy 10 days after inoculation and were removed to the greenhouse. After 2 weeks in the greenhouse only 10 plants were still free of disease symptoms. Leaves of these plants remained free of anthracnose symptoms; however, within 30 days after inoculation all plants developed stem lesions at the soil line and died. Twenty-five seeds of the other citron line, W695, were obtained from Dr. J. R. Wall, United States Department of Agriculture Vegetable Breeding Laboratory, Charleston, South Carolina. It originally was obtained from W. Unkles, Montana Nurseries, Union of South Africa and was reported by Goode (9) as resistant in preliminary tests. Twenty seedlings of this citron were inoculated. Ten plants were susceptible and 10 plants showed little or no foliage symptoms; however, these plants eventually developed anthracnose lesions at the soil line and died. One plant showed no symptoms for about 2 months but then a lesion developed on the base of the stem near the soil line. A graft of the apical portion of this plant was made

Table 1. Commercial watermelon varieties susceptible to races 1, 2, and 3 of C. lagenarium.

Varieties tested[a]		
Black Diamond	Honey Cream	Norman Parker
Blacklee	Ice Cream	Northern Sweet
Blue Watson	Improved Georgia Rattlesnake	Preserving Citron
Blue Wonder	Irish Gray	Purdue Hawkesbury
California Honey	Ironsides	Red Heart Stone Mountain
Cannon Ball Special	Kleckley No. 6	Rhode Island Red
Chilean Black Seeded	Kleckley Sweet	Snyder
Citron	Kleckley Sweet Improved	Stone Mountain
Cletex	Kleckley Sweet Wondermelon	Stone Mountain No. 5
Cole's Early	Klondike	Striped Klondike
Colorado Preserving Citron	Klondike Black Seeded	Sugar Baby
Cut Red Watson	Klondike R-7	Sunnybrook Hybrid
Darlington	Klondike Striped Blue Ribbon	Super Black Diamond (Yellow Belly Strain)
Dixie Queen	Klondike Striped Wilt Resistant	Super Red Heart Stone Mountain
Dixie Queen Wilt Resistant	Ledman	Sweetheart
Duke Creek	Leesburg	Takii Gem
Early Canada	Long Lucuous	Tendersweet
Early Kansas	Golden Honey	Texas Yellow Meat
Ferry's Favorite	Miles	Thurmond Gray
Florida Giant	Monte Cristo	Tom Watson
Fordhook Early	Mountain Hoosier	Wilt Resistant Dixie Queen
Garrison	Nancy Hawks	Winona
Georgia Rattlesnake	New Hampshire Midget	Winter Queen
Golden Honey		Wonder
Graystone		Yellow Belly Black Diamond
Halbert's Honey		
Harris' Earliest		
Hawkesbury		

[a]Seed for these tests were furnished by the following: Associated Seed Growers, Inc.; W. Atlee Burpee Company; Corneli Seed Company; Ferry-Morse Seed Company; Joseph Harris Company, Inc.; The Kilgore Seed Company; Simpson Nursery Company; T. W. Wood & Son.

Table 2. Varieties and advanced breeding lines resistant to races 1 and 3 and susceptible to race 2 of C. lagenarium.

Variety or breeding line	
Black Kleckley	Georgia No. 4[a]
Blackstone	Hope Diamond[b]
Charleston Gray	N.C. 20[c]
Congo	N.C. 21[c]
Fairfax	N.C. 22[c]

[a]Advanced breeding line; seed furnished by E. S. Luttrell, Georgia Experiment Station, Experiment, Georgia.

[b]New variety; seed furnished by V. M. Watts, University of Arkansas, Fayetteville, Arkansas.

[c]Advanced breeding lines; midget type watermelons resistant to wilt and anthracnose, North Carolina Experiment Station, Raleigh, North Carolina.

Table 3. Citrullus plant introductions susceptible to race 2 of C. lagenarium.

Plant Introduction Number[a]		
161373-162667	172787 through 172805	183217-183218
163202 through 163205	173667 through 173670	183299-183300
163572-163574	173888-174089	183398-183399
164146-164247	174099 through 174101	183673-184800
164248-164460	174104 through 174109	185030-185635
164474-164475	174812-175102	185636-186489
164539-164543	175650 through 175653	186490-186973
164550-164570	175655-175657	186974-187317
164633-164634	175658 through 175665	188808-189225
164636-164369	176485 through 176499	189316-189318
164655-164665	176905 through 176919	190050-192937
164685-164687	176921 through 176923	192938-193490
164708-164709	177318 through 177331	193963 through 193965
164737-164748	178870 through 178877	195562-195771
164804-164977	179232 through 179243	195927-195928
164992-164998	179660 through 179662	197416-200732
165002-165024	179875 through 179886	203551-204689
165448-165449	180275 through 180278	207473-208740 W
165451-165523	180436-180427	208740 B-210017
166993-167026	181740 through 181744	211011-211849
167045-167059	181935 through 181938	211850 through 211852
167124 through 167126	182175 through 182177	212208-212209 W
167129-167222	182179 through 182181	212209 B-212287
169232 through 169297	182183-182932	212288-212289
169299 -169300	182933 through 182935	212316-212939
171392-171579	183022-183023	212983-217937
171580 through 171587	183033-183123	217938-219691
172341-172786	183124 through 183126	222731-222714

[a]Seed of these Plant Introductions were furnished by Dr. Edwin James, Southern Regional Plant Introduction Station, Experiment, Georgia. The dash (-) indicates only that the two lines listed were tested, for example, 161373-162667. The word "through" is used to indicate that lines not listed were also tested, for example, 163202 through 163205 indicates that 163203 and 163204 were also tested.

onto a Charleston Gray root stock and subsequently this plant was used as the pollen parent in crosses with other breeding lines. In addition selfed seed were obtained. Approximately 3/4 of the selfed progeny appeared to be resistant. The progeny of W695, classed as resistant when inoculated with race 2, show little or no foliage infection, however, such plants developed lesions at the soil line within 1 week to 2 months after inoculation. Such lesions have always been lethal. In a field test in 1957, approximately 3/4 of the plants of this line were resistant to race 2. Charleston Gray plants in adjacent rows were all severely affected. Plants of W695 were extremely susceptible under field conditions to both Mycosphaerella melonis (Pass.) Chiu & J. C. Walker and to a similar fungus, Ascochyta sp.

Seedlings of line W695 are also resistant to races 1 and 3. The level of resistance to these two races appears to be similar to that of Charleston Gray.

Inheritance Studies

In studying the inheritance of resistance to races 1 and 3, the varieties and lines used were N.C. 9-2 (susceptible), N.C. 11 (susceptible), New Hampshire Midget (susceptible), and Charleston Gray (resistant). Populations tested were: 1) the susceptible and resistant parents; 2) F_1 progeny of the crosses N.C. 11 x Charleston Gray and New Hampshire Midget x Charleston Gray; 3) F_2 progeny of the cross N.C. 9-2 x Charleston Gray; and 4) the backcross progeny (N.C. 9-2 x Charleston Gray) x Charleston Gray, (N.C. 11 x Charleston Gray) x N.C. 11, (New Hampshire Midget x Charleston Gray) x New Hampshire Midget and (N.C. 9-2 x Charleston Gray) x N.C. 9-2.

Table 4. Ratio of resistant to susceptible plants of F_2 and F_1 backcrossed to the susceptible parent and to the resistant parent when inoculated with C. lagenarium races 1 and 3.

Population	Resistant	Susceptible	Expected ratio	P Value
		Inoculations with race 1		
Parents				
N.C. 9-2	1	26	0:27	
N.C. 11	0	37	0:37	
New Hampshire Midget	2	36	0:38	
Charleston Gray	31	2	33:0	
W695[a]	20	0	20:0	
F_1				
N.C. 11 x Charleston Gray	46	4	50:0	
New Hampshire Midget x Charleston Gray	27	2	29:0	
F_2				
N.C. 9-2 x Charleston Gray	44	16	3:1	.70 to .80
Backcrosses				
(N.C. 9-2 x Charleston Gray) x N.C. 9-2	59	67	1:1	.30 to .50
(N.C. 11 x Charleston Gray) x N.C. 11	46	51	1:1	.50 to.70
(New Hampshire Midget x Charleston Gray) x New Hampshire Midget	36	36	1:1	.99 to 1.0
(N.C. 9-2 x Charleston Gray) x Charleston Gray	46	4	50:0	
		Inoculations with race 3		
Parents				
N.C. 9-2	5	30	0:35	
N.C. 11	2	22	0:24	
New Hampshire Midget	6	29	0:35	
Charleston Gray	27	0	27:0	
W695[a]	22	9	3:1	
F_1				
N.C. 11 x Charleston Gray	45	3	48:0	
F_2				
N.C. 9-2 x Charleston Gray	43	18	3:1	.30 to .50
Backcrosses				
(N.C. 9-2 x Charleston Gray) x N.C. 9-2	83	85	1:1	.80 to .90
(N.C. 11 x Charleston Gray) x N.C. 11	52	52	1:1	.99 to 1.0
(New Hampshire Midget x Charleston Gray) x New Hampshire Midget	38	41	1:1	.70 to .80
(N.C. 9-2 x Charleston Gray) x Charleston Gray	42	1	43:0	

[a]Seed of the two populations of W695 came from different plants. The line segregating for resistance to race 3 also segregated in the field for flesh color and fruit characteristics. Hence, this line was probably the F_2 of an outcross.

The monogenic resistance hypothesis was subjected to the Chi square test. A summary of the Chi square test is presented in Table 4. A very close fit to the 3:1 and 1:1 ratio of each line against races 1 and 3 demonstrates that resistance to each is monofactorial. Since all varieties resistant to race 1 were resistant to race 3 and vice versa, and the inheritance of each was monofactorial and dominant, it appeared that resistance to both races was governed by the same gene. To verify this, all plants classed as resistant to races 1 and 3 were placed in the greenhouse for 1 week then reinoculated. Plants previously shown to be resistant to race 1 were inoculated with race 3 and vice versa (Table 5). Very few plants which had been classed as resistant to race 1 were affected when inoculated with race 3; plants resistant to race 3 were also resistant to race 1. The proportion of reinoculated plants which became diseased was no greater than that of Charleston Gray, the resistant parent. These results further verify that resistance to both races is governed by the same gene.

Table 5. Reactions of watermelon plants previously inoculated and classed as resistant and then reinoculated with race 1 or race 3[a].

Line	Plants resistant to race 3 and reinoculated with race 1			Plants resistant to race 1 and reinoculated with race 3		
		Number of plants			Number of plants	
	Total	Resistant	Susceptible	Total	Resistant	Susceptible
N. C. 9-2	1	0	1	-	-	-
N. C. 11	4	0	4	-	-	-
New Hampshire Midget	5	0	5	-	-	-
Charleston Gray	20	18	2	21	21	0
F_1						
N. C. 11 x Charleston Gray	16	16	0	33	32	1
New Hampshire Midget x Charleston Gray	5	4	1	20	20	0
F_2						
N. C. 9-2 x Charleston Gray	17	16	1	23	22	1
Backcrosses (N. C. 9-2 x Charleston Gray) x Charleston Gray	25	24	1	21	21	0
(N. C. 9-2 x Charleston Gray) x N. C. 9-2	27	24	3	23	23	0
(N. C. 11 x Charleston Gray) x N. C. 11	19	18	1	19	18	1
(New Hampshire Midget x Charleston Gray) x New Hampshire Midget	20	19	1	21	21	0

[a]Lines inoculated previously with and classed as resistant to race 1 were inoculated with race 3 or vice versa.

Since preliminary results had suggested that resistance to race 2 was also monogenic and dominant, F_1, F_2 and backcross progeny of crosses between Charleston Gray and W695 were also made. Two inoculations were made. In the first test most populations fitted the expected 3:1 and 1:1 ratios; however, in the second test no such results were obtained. The results of these 2 tests are shown in Table 6.

DISCUSSION

In inoculations with races 1 and 3 very few plants gave reactions which were unexpected. When plants were inoculated under field conditions resistance to races 1 and 3 approached immunity under North Carolina conditions. However, plants of resistant varieties usually showed

Table 6. Results of inoculations with C. lagenarium race 2 to W695, Charleston Gray and to various progeny of crosses between these two lines.

Populations	Test 1			Test 2		
	Total	Resistant	Susceptible	Total	Resistant	Susceptible
W695	52	39	13	170	57	113
Charleston Gray	44	7	37	83	21	62
F_1						
W695 x Charleston Gray	36	33	3	26	1	25
F_2						
W695 x Charleston Gray	283	225	58	255	91	164
F_1 x Charleston Gray	29	17	12	102	25	78
F_1 x W695	63	49	14	220	65	155

a few leaf lesions in seedling inoculation tests when the procedures outlined by Goode (9) were followed. Furthermore, a few seedlings of resistant varieties were killed in each test. Occasionally, as indicated in Table 5, a susceptible plant may have also escaped infection and have been classed as resistant. In the test reported herein susceptible and resistant were included in each inoculation test. The reactions of the segregating populations did not differ statistically from the expected ratios and were also consistent with the results obtained from inoculated plants of resistant and susceptible varieties. The single dominant factor reported by Layton (11) for anthracnose resistance governs resistance to C. lagenarium races 1 and 3.

The 19 Plant Introductions previously reported by Goode as resistant or tolerant to race 2 did not appear resistant in these tests.

The level of resistance to race 2 in W695, a citron, appeared to be satisfactory for watermelon production under field conditions. Although no lesions have been observed on fruits of resistant W695 plants, a few small leaf spots and small stem lesions were observed in 1957. In this test, however, a row of W695 plants were grown between two rows of Charleston Gray plants. The Charleston Gray plants in this test were completely killed by C. lagenarium race 2.

The level of resistance to race 2 in W695 also appeared to be less complete in seedling inoculation tests than the resistance to races 1 and 3 carried by varieties such as Charleston Gray. In seedling tests plants that showed no leaf lesions frequently developed a single lethal stem lesion.

While the techniques used in evaluating progeny for resistance to races 1 and 3 may not be perfect, repeatable results can be obtained from test to test. As is indicated in Table 6, repeatable results have been difficult to obtain with progeny segregating for resistance to race 2. Although resistance to race 2 may also be controlled by a single factor pair, refinements in inoculation techniques appear to be necessary before the mode of inheritance can be demonstrated.

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REACTION OF RYE VARIETIES TO LEAF RUST[1]

Darrell D. Morey[2]

Chapman et al. (1) have shown that Gator rye contains a high percentage of plants resistant to leaf rust. Wells (2) reported that Explorer rye has considerable resistance to leaf rust. Discovery of other domestic or foreign varieties with resistance to leaf rust would be an asset to the rye program, especially in the southeastern States, where leaf rust is a major problem.

Seventy-five foreign rye varieties or selections and 31 North American varieties or selections were tested for susceptibility to a local collection of leaf rust, Puccinia recondita Rob. ex Desm., at Tifton, Georgia. Results represent the average of three greenhouse tests in which 12 to 15 seedlings per pot were inoculated.

Among the foreign ryes, only six exhibited any seedling resistance to leaf rust (Table 1). Sixty-nine rye varieties obtained from France, Morocco, Egypt, South Africa, Chile, Argentina, Uruguay, India, Russia, and Australia were completely susceptible to leaf rust and are not listed.

Table 1. Reaction of foreign rye varieties or selections to leaf rust of rye at Tifton, Georgia, 1959.

Variety or selection	Percent of plants resistant	Source
Sel. MF 4F-F3	.25	Estacion Experimental
Liho (Castelar) P.C.F. 33	.15	Pergamino, F.C.N.G.B.N. Pergamino, Argentina
Selecta No. C 155/55	.20	Instituto de Fitotecnia Castelar, Argentina
C. 58/85 W.R. 174	.40	Div. of Crops and Pastures Pretoria, South Africa
Kenya rye	.10	Plant Breeding Station Kenya Colony, Njoro, Kenya, Africa
Seigle No. 244	.20	Service de 1'Experimentation Agricole Maison-Carree, Algeria

No varieties except lines selected from Gator approached the resistance of Gator. Three experimental synthetic varieties bred from Gator at the North Florida Experiment Station, Quincy, Florida varied in rust resistance, but one appeared very promising. Hazel, an experimental rye obtained from Gator by recurrent selection for larger seed size, apparently has retained the full leaf-rust resistance of its Gator parent (Table 2).

If large populations of Florida Black, Mississippi Abruzzi, Wren's Abruzzi, and possibly other rye varieties are subjected to leaf rust, an occasional resistant plant can be found. Thus five selections from the Mississippi Experiment Station derived from Mississippi Abruzzi show a significant increase in resistance to leaf rust. Because of cross-pollination, sterility factors, and the heterozygous nature of rye, breeding and selecting for disease resistance

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Table 2. Reaction of North American rye varieties or selections to rye leaf rust at Tifton, Georgia, 1959.

Variety or selection	Percent of plants resistant	Source
Gator	.70	Florida and Georgia
Hazel (Experimental)	.70	Georgia
Quincy Synthetic 1	.15	Florida
Quincy Synthetic 2	.73	Florida
Quincy Synthetic 3	.25	Florida
Explorer	.30	Mississippi
Mississippi Synthetic 4	.20	Mississippi
Mississippi Synthetic 5	.17	Mississippi
Mississippi Synthetic 6	.17	Mississippi
Mississippi Abruzzi	.14	Mississippi
Florida Black	.00	Florida
Florida Black, Gainesville selection	.00	Florida
French 492	.00	Georgia
Elbon	.00	Oklahoma
Wren's Abruzzi	.00	Georgia
Wood's Abruzzi	.00	Virginia
Prolific Spring	.00	Montana
Merced Spring	.00	California
Coward's Abruzzi	.00	South Carolina
Adams	.00	Wisconsin
Pierre	.00	South Dakota
Balbo	.00	Michigan
Rosen	.00	Tennessee
Antelope	.00	Saskatchewan, Canada
Norton	.00	Minnesota
Caribou	.00	Minnesota
Emerald	.00	Minnesota
C.D. 5169	.00	Minnesota
Kings II	.00	Minnesota
Selection 2	.00	Minnesota
Tetra Petkus (check)	.00	Pennsylvania

become more difficult. It has been possible, however, to obtain varieties with moderate to good resistance to a local collection of leaf rust of rye (Table 2).

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CHISEL APPLICATION OF METHYL BROMIDE FOR CONTROL OF ROOT-KNOT NEMATODE AND FUSARIUM WILT

Ivan J. Thomason[1]

Summary

When injected into the soil with chisel applicator and covered with tarps, methyl bromide at 200 and 300 pounds per acre controlled the root-knot nematode and the Fusarium wilt organism to a depth of 3 feet. A dosage of 300 pounds per acre of methyl bromide controlled Sclerotium bataticola, but not Rhizoctonia solani and a Stemphyllium sp.

Chisel application of methyl bromide followed by covering with polyethylene tarps has made possible the economic use of this fumigant on large areas. In the Coachella Valley of southern California 335 acres of land used for the production of tomato transplants was treated with methyl bromide for control of broomrape, Orobanche ludoviciana var. Cooperi, weeds, and root-knot nematodes in 1958.

The increased use of methyl bromide on large areas has resulted from the more uniform distribution and greater efficiency obtained when this fumigant is injected and covered with tarps.

Sher, Thomason and McCaslin in 1958[2] reported a high level of nematode control by chisel applications of 150 to 200 pounds per acre of methyl bromide. However, further information regarding nematode, fungus, and weed control was needed. Also, control of a wide range of soil-borne pests would help to justify the cost of treatment, which at present appears to be limited to special high net return crops.

A test was conducted at Mira Loma, California on land planted to blackeye beans, Vigna sinensis, to determine if the root-knot nematode, Meloidogyne javanica, and the Fusarium wilt fungus, Fusarium oxysporum f. tracheiphilum, could be controlled by dosages of 200 and 300 pounds per acre of methyl bromide. The beans were used essentially as an assay crop for the two parasites and would not support the cost of fumigation.

PROCEDURE

The test consisted of the following four treatments: 1) methyl bromide (Weedfume[3]) 200 pounds per acre, 2) methyl bromide 300 pounds per acre, 3) ethylene dibromide (EDB W-85) 86.5 pounds per acre, and 4) non-treated control. Individual plots were 18 x 60 feet and replicated three times. The field had a crop of barley during the winter of 1957-58 and the stubble had just been plowed under prior to irrigating. Considerable undecomposed barley stubble was present in the soil. The field was sprinkler irrigated on May 17 and treatments were applied May 19, 1958.

The soil was a loamy sand, the subsoil being somewhat higher in clay content. A mechanical analysis of soil samples from these depths gave the following results: 0-1 foot depth -- clay 2.9 percent, silt 9.6 percent, sand 87.5 percent; 1-2 foot depth -- clay 3.9 percent, silt 8.7 percent, sand 87.4 percent; and 2-3 foot depth -- clay 5.2 percent, silt 7.4 percent, sand 87.4 percent.

The methyl bromide was applied at a depth of 8 inches with shanks on 10-inch centers with a chisel type applicator. Tarps were placed over the treated soil within 15 minutes after application and remained on for 22 hours. Ethylene dibromide was injected at a depth of 7 inches with shanks on 12-inch centers and then the soil was firmed with a drag. Blackeye beans were

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[2]Sher, S. A., Ivan J. Thomason, and R. L. McCaslin. 1958. Chisel application of methyl bromide for root-knot nematode control. Plant Disease Reptr. 42: 288-290.

[3]Weedfume (70% methyl bromide and 30% petroleum). Supplied and applied by R. L. McCaslin, Entomologist, Neil A. Mclean Co., Los Angeles, California.

seeded in all plots on May 21. Alternate rows of Grant (nematode susceptible) and Chino 3 (wilt susceptible) beans were planted in each plot.

Soil samples were taken with a soil tube on May 23 to avoid possible contamination of treated plots by cultivating equipment during the growing season. Three replicates each of the check and two methyl bromide treatments were sampled. Six cores from each of three depths (0-1 foot, 1-2 foot, and 2-3 foot) in each replicate were obtained, bulked, and planted to Chino 3 blackeye in the greenhouse. At the end of 2 months these beans were examined for the presence of nematode galls on the roots and discoloration in the vascular system of the stem. Isolations were made from stem sections obtained from below the cotyledonary node for the presence of fungi.

Ten plants of each variety of bean in each plot were scored for amount of root-knot nematode galling, vascular discoloration, and cortical rot at harvest time. Fungus isolations were made from cortical and vascular tissues of these plants.

RESULTS

Plants Grown in Soil Samples taken 4 Days after Treating

Examination of the plants grown in the soil samples indicated that treatment with methyl bromide at 200 and 300 pounds per acre resulted in control of root-knot nematodes to a depth of 3 feet (Table 1). These same treatments controlled the Fusarium wilt fungus. One plant growing in soil from the 2-3 foot depth of one of the plots which had been treated with 200 pounds per acre of methyl bromide showed a trace of vascular discoloration. However, Fusarium oxysporum was not isolated from this plant (Table 1).

Table 1. Root galling, vascular discoloration, and fungi isolated from blackeye beans (var. Chino 3) grown in soil samples from non-treated and methyl bromide treated plots[a].

Treatment	Depth of sample (in feet)	Root galling	Vascular discoloration	Fungus: Rhizoctonia solani	Fusarium oxysporum	Fusarium roseum	Sclerotium bataticola	Pythium sp.
None	0-1	3[b]	3	3	2	0	1	1
	1-2	3	1	2	2	0	1	1
	2-3	2	1	1	2	0	0	0
Methyl bromide 200 lbs./ac.	0-1	0	0	1	0	1	2	0
	1-2	0	0	1	0	2	3	0
	2-3	0	1	2	0	2	0	0
Methyl bromide 300 lbs./ac.	0-1	0	0	1	0	2	0	0
	1-2	0	0	1	0	2	0	0
	2-3	0	0	2	0	2	0	0

[a]Methyl bromide applied May 19, 1958, soil samples obtained and planted to beans May 23, 1958, roots examined and stem sections plated for fungus isolations July 29, 1958.

[b]Figures in table indicate number of times root galling, vascular discoloration, and/or a fungus was seen or isolated from three plants (one plant in each of three replicates).

Rhizoctonia solani was isolated from plants grown in both treated and non-treated soil. Sclerotium bataticola (Macrophomina phaseoli) was recovered from plants grown in the non-treated soil and the soil treated with 200 pounds per acre of methyl bromide. It was not recovered from plants grown in soil treated with 300 pounds per acre of methyl bromide. Pythium was isolated only from plants grown in non-treated soil. Treating the soil with methyl bromide resulted in the isolation of a species of Fusarium, apparently F. roseum, from plants in the treated soil but not from those in non-treated soil.

Examination of Field Grown Plants

Bean plants grown on soil treated with 200 pounds per acre of methyl bromide were free from nematode galling at the end of the growing season (Table 2). All plants grown on soil

Table 2. Root galling, vascular discoloration, and cortical rot indexes on blackeye beans (var. Grant and Chino 3) on methyl bromide fumigated soil.

Treatment	Dosage lbs./ac.	Root galling[a] (0-4)	Vascular discoloration[b] (0-5)	Cortical rot[c] (0-5)
Untreated	---	3.3	1.7	2.1
Methyl bromide	200	0	0.3	0.6
Methyl bromide	300	0.1	0.5	0.8
EDB W-85	86	0.8	1.3	2.0

[a]0 = Roots clean; 1 = 1-25% of roots galled; 2 = 26-50% of roots galled; 3 = 51-75% of roots galled; 4 = 76-100% of roots galled.
[b]Vascular necrosis or discoloration was used as an index of the severity of Fusarium wilt. Scored as follows: 0 = no xylem necrosis, 1 = trace, 2 = 25%, 3 = 50%, 4 = 75% of xylem necrotic, and 5 = plants dead.
[c]Cortical tissue of the stem below the cotyledonary node scored for severity of necrosis as indicated above for vascular discoloration.

Table 3. Fungi isolated from cortical and vascular tissue of field grown bean plants (variety Chino 3).

Treatment	Tissue	Fungus: Fusarium oxysporum	Sclerotium bataticola	Rhizoctonia solani	Stemphyllium sp.
None	Vascular	12/14[a]	7/14	2/14	0/14
	Cortical	8/8	4/8	0/8	0/8
Methyl bromide 200 lbs./ac.	Vascular	0/12	1/12	1/12	0/12
	Cortical	0/6	1/6	2/6	2/6
Methyl bromide 300 lbs./ac.	Vascular	2/12	1/12	0/12	1/12
	Cortical	1/6	0/6	4/6	4/6

[a]Numerator gives the number of times the fungus was isolated from the number of stem sections given in the denominator.

treated with 300 pounds per acre of methyl bromide were free of galling except for three plants in one replication which showed a trace of galling. Nematodes were not recovered from soil samples taken from this treatment prior to planting. The galling may have resulted from contamination of this plot by cultivating equipment moving from an adjacent non-treated plot.

Vascular discoloration was observed in plants from all treatments (Table 2). However, only a trace of xylem necrosis was found in a few plants of the many grown in soil treated with 200 and 300 pounds per acre of methyl bromide. This was true for cortical rot of the stem also (Table 2). F. oxysporum f. tracheiphilum was not isolated from vascular tissue of plants grown in soil treated with 200 pounds per acre of methyl bromide (Table 3). As with the root-knot nematode, a few plants in one replicate of the 300 pounds per acre-treatment showed a trace of wilt and the fungus was recovered from the stem sections. Roots of plants grown in

the non-treated soil were severely galled and stems showed a moderate amount of vascular discoloration. Fusarium oxysporum was isolated from 12 of 14 plants from the non-treated soil. Sclerotium bataticola apparently was controlled by 300 pounds per acre of methyl bromide. Rhizoctonia solani was isolated from the cortical tissue of plants grown in all treatments[4].

REMARKS

Early in the growing season stands on the methyl bromide treated plots were satisfactory but the plants showed severe stunting. Presumably, this was due to the depression of nitrifying bacteria in the soil by methyl bromide and the accumulation of ammonia. Later in the growing season the plants recovered and surpassed all plants in other treated areas in growth. EDB controlled the root-knot nematodes and growth of the bean plants was much better on EDB treated soil than on non-treated soil. However, the bean plants in the EDB treated plots matured early before soil moisture was depleted, indicating that fungi isolated from the stems were seriously injuring the plants. The bean plants on the methyl bromide treated plots were still green and in bloom at the time the grower cut the plants in preparation for harvest.

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[4]The author wishes to express his appreciation to Drs. Peter Tsao and D. C. Erwin, Department of Plant Pathology, Citrus Experiment Station, Riverside, California, for aid in isolating and identifying the fungi.

INFLUENCE OF SOIL TEMPERATURE ON VERTICILLIUM HADROMYCOSIS OF COTTON IN CALIFORNIA[1]

P. M. Halisky, R. H. Garber, and W. C. Schnathorst

Abstract

Greenhouse and laboratory experiments were conducted to determine the effect of soil temperature on Verticillium wilt of cotton. Soil temperatures were maintained at 15°, 20°, 25°, 30° and 35°C. Deltapine 15, a wilt-susceptible variety of cotton, was inoculated with Verticillium albo-atrum Reinke & Berth. and grown in replicated trials at these temperatures. The soil temperatures most favorable for disease development were 20° and 25°. No wilt symptoms developed at a constant soil temperature of 35°. Growth studies were made with the fungus at temperatures ranging from 12° to 39°C at 3° intervals. Growth was maximum at 21° and 24° and no measurable growth occurred above 30°. The influence of soil temperature on wilt development in cotton apparently is directly related to the effect of temperature on the growth and activity of the pathogen.

The occurrence, widespread distribution, and economic importance of Verticillium hadromycosis of crop plants in California is more widely recognized every year (3, 5, 12). Cotton is the leading farm crop in the State and Verticillium wilt is its most destructive disease. Estimates compiled by the Cotton Disease Council show losses from Verticillium wilt in California to be 13.0, 4.5, 2.1, 2.0, and 3.2 percent, respectively, for the years 1953 through 1957 -- an average yield reduction of 4.96 percent annually. Verticillium wilt may also seriously affect the quality of cotton lint, thus increasing the loss to growers. In California, cotton is grown in six counties in the San Joaquin Valley and in two counties in the Imperial Valley. Much of the cotton in the San Joaquin Valley is affected by Verticillium wilt but the disease has not been reported from the Imperial Valley of this State. The reason remains unknown. Disease expression and wilt severity may vary greatly in a cotton field from year to year. A better understanding of environmental influences upon disease development might lead to control recommendations of practical value to growers.

GREENHOUSE EXPERIMENTS

Verticillium hadromycosis of cotton is generally considered a cool-temperature disease. A series of experiments was conducted to determine the extent of this relationship under controlled experimental conditions. Studies with Verticillium wilt of cotton were made in the greenhouse in specially constructed constant-temperature tanks. The temperature in each tank was maintained with circulating water. Seedlings of Deltapine 15, a wilt-susceptible variety of cotton, were inoculated by immersing the roots in a spore suspension of Verticillium albo-atrum. Inoculated seedlings were transplanted into steam-sterilized Yolo fine sandy loam in partly submerged two-gallon crocks. The temperatures of the circulating water in the four tanks were maintained at 15°, 20°, 25° and 30°C. Four consecutive trials were conducted, each consisting of 8 to 11 replicated crocks containing six to nine cotton seedlings. The foliage was exposed to the same air temperature which increased during the trials from 23° in February to a maximum of 29° in July and then gradually decreased to 23° in November. The number of infected seedlings was ascertained from careful examination for both foliar symptoms and vascular discoloration. The latter criterion, especially useful in revealing incipient infections, was generally more reliable. The disease readings were made about 2 months after inoculation, and the data converted to infection percentages.

The choice of a representative isolate of V. albo-atrum for these studies was considered

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[1] Contribution from the Agricultural Experiment Station, University of California, Davis, California, and Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

important. Not only is the fungus characterized by genetic variation in culture, but the distinct types differ significantly in pathogenicity (8, 10, 11). The wild type of isolate (Fig. 1) used in these studies was selected because it was consistently isolated from wilted cotton by Garber (3) during 5 years of investigation of this disease in California.

The comparative effects of controlled soil temperature on the extent of infection of cotton by the Verticillium wilt fungus are shown in Table 1. The data presented are the average percentages of infection in all the replications in each trial at the temperature indicated. Each

Table 1. Effect of constant soil temperatures on disease development in Deltapine 15 cotton infected by Verticillium albo-atrum.

Trials	Replications	Soil temperature and disease percentage			
		15°C	20°C	25°C	30°C
1. (February-March)	8[a]	52	98	95	81
2. (April-May)	11	74	79	91	62
3. (June-July)	11	49	63	70	23
4. (October-November)	12	-[b]	60	82	70
Average Percentage		58	75	85	59

[a]Each replicate consisted of six to nine cotton seedlings.

[b]The temperature in this tank was raised to 35°C in trial 4.
No disease symptoms developed at this relatively high soil temperature.

percentage figure thus represents the average disease reaction of about 72 cotton plants. The data in the table indicate that soil temperatures of 20° and 25°C are more favorable for wilt development in cotton and higher temperatures are less favorable. When the temperature of the soil was elevated to 35°, wilt development was completely suppressed. The lowest average infections were obtained in midsummer (trial 3), when greenhouse air temperatures were highest.

LABORATORY INVESTIGATIONS

Studies were conducted with V. albo-atrum to relate its growth activity to greenhouse infection studies. The same isolate was used in both studies. Cultures of the fungus were grown on potato-dextrose agar (PDA) at constant temperatures ranging from 12° to 39°C at 3° intervals. V. albo-atrum is known to grow relatively slowly in artificial culture. After 14 days of incubation the average colony diameters of seven replicated cultures were determined. Representative cultures are shown in Figure 1. On the basis of colony-diameter determinations, 21° and 24° were the most favorable temperatures for the growth of the fungus in culture. Respective average diameters after 14 days of incubation were 46 and 47 mm. The temperature range for the growth of V. albo-atrum in culture was shown by Wilhelm (13) to lie between 10° and 31°. The present results are in agreement with Wilhelm's observations, since no measurable growth occurred at 33°, 36°, and 39°. Furthermore, more microsclerotia were formed at the lower temperatures than at the higher temperatures. The colonies were white at 30° and jet-black at 12° and 15°. It is apparent from these and other studies that higher temperatures impart a direct inhibitory effect on the growth activity and on the parasitic behavior of V. albo-atrum.

DISCUSSION

The experimental data obtained in these studies indicate that the most favorable soil temperatures for the development of Verticillium wilt of cotton are 20° and 25°C. These results are comparable with those obtained by other investigators (2, 4, 5, 6, 7, 8) studying Verticillium wilt of tomato, pepper, cotton, eggplant, potato and peppermint.

The notable absence of Verticillium wilt from the Imperial Valley of California has often been attributed to high soil temperatures which are prohibitive to the growth and development of the pathogen. Soil temperature data collected from cotton fields in southern California, however, do not support this explanation. During the months of March, April and May the soil temperatures at depths of 3 to 36 inches were found to be optimal for the growth of V. albo-atrum according to the results of this study. In the hot summer months of June, July, August

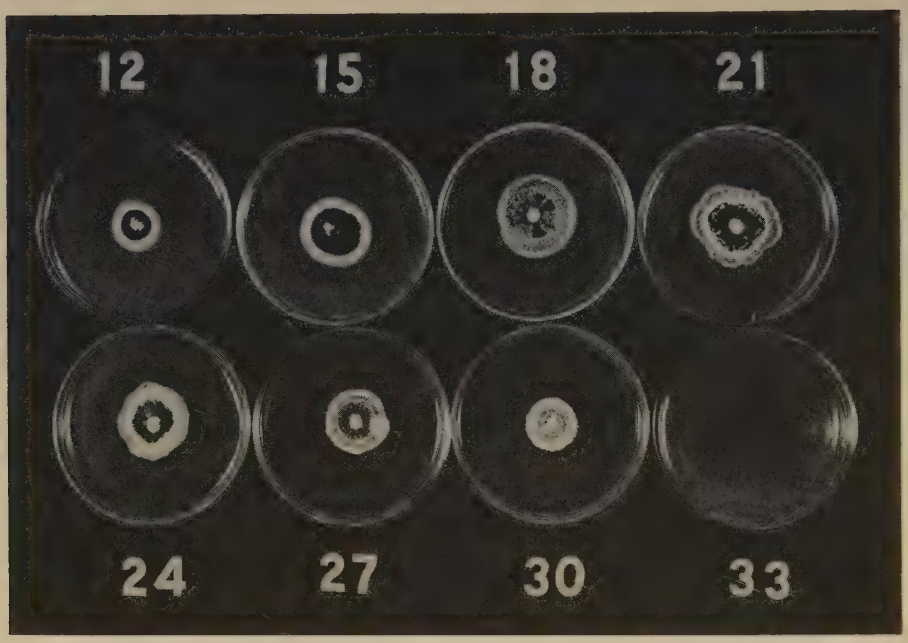

FIGURE 1. Cultures of Verticillium albo-atrum after 14 days at the temperatures (^{o}C) indicated.

and September soil temperatures at depths of 12 to 36 inches remained below 30^{o}C. In greenhouse studies considerable wilt (59 percent) developed at this temperature. Although an incubation temperature of 33^{o} suppressed the growth of the pathogen (Fig. 1) and 35^{o} completely inhibited disease development (Table 1), no evidence was found that soil temperatures of this magnitude exist at root level in cotton fields. Apparently factors other than soil temperature are responsible for the erratic distribution of Verticillium wilt of cotton in California.

The influence of air temperature on the development of Verticillium wilt in cotton has received little attention. Edgington and Walker (2) found that both air and soil temperatures were important factors in the development of Verticillium wilt of tomato. The partial effect of air temperature on wilt development in cotton was observed in this study. The lowest average infections were obtained in midsummer when greenhouse air temperatures were highest. The greatest reduction in percentage of infected plants occurred in combination with the highest soil temperature. Daily air temperatures in the warmer cotton-growing valleys of California frequently average 32^{o} C during the summer months. High air temperatures may directly influence host physiology and alter its resistance or susceptibility to Verticillium wilt. Since microsclerotia are more abundant in host cortex and stem-pith tissues above ground, the inhibitory influence of high air temperatures on the numbers of microsclerotia formed should not be overlooked. A failure of microsclerotial formation would account for a reduction in the inoculum potential and a subsequent decrease in disease severity.

The role of microsclerotia of V. albo-atrum is of paramount importance both in fungus survival and in direct relation to pathogenicity. Several studies (8, 10, 11) have revealed that virulence is apparently correlated with microsclerotial formation and that pronounced loss of pathogenicity is related to the loss of the sclerotial type in culture. The relation of temperature to microsclerotial survival was studied by Nelson and Wilhelm (9). In the dry stage microsclerotia of V. albo-atrum were found capable of surviving many months at 34^{o}, 37^{o}, 40^{o} and 49^{o} to 50^{o}C. When immersed in water, however, complete kill within 40 minutes was obtained at 47^{o}, 48^{o}, 49^{o}, and 50^{o}. These results suggest that in the dry state microsclerotia could readily survive in the warmest cotton-growing areas of California. Since the presence of moisture shortens the period of viability of the microsclerotia, their longevity under field conditions may be curtailed by irrigation. The combined influence of soil moisture and temperature may affect the distribution of Verticillium wilt in California.

The relation of temperature to the growth and parasitic activity of V. albo-atrum is of primary importance and is accordingly emphasized in these studies. The growth record of the fungus in culture compared with greenhouse infection data indicates a direct relationship.

Higher temperatures inhibited microsclerotial formation and suppressed mycelial development. The physiologic processes governing microsclerotial formation are apparently also related to the effect of temperature. Becker (1) found that microsclerotial production in V. albo-atrum was directly correlated with polyphenolase activity in the fungus mycelium. He observed maximal enzymatic activity at 25°C whereas at 30° polyphenolase activity was insignificant. These results help to explain the differences in microsclerotial formation at different temperatures (Fig. 1) and indicate the restrictive effect of temperature on the growth, development, and pathogenicity of V. albo-atrum.

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THREE FACTORS WHICH INFLUENCE THE LOCAL SPREAD OF OAK WILT IN FIVE NORTHEASTERN COUNTIES OF WEST VIRGINIA[1]

W. H. Gillespie and R. P. True[2]

Abstract

Local spread of oak wilt is favored by shallow soils, the availability of compatible[3] oaks, and the number of dead and currently wilting trees at the infection center when found and treated in northeastern West Virginia.

INTRODUCTION

Few observations have been reported in regard to the influence of environmental factors upon the local spread of oak wilt[4]. It was reported by Henry et al. in 1944 (6) and again by Kuntz and Riker in 1950 (8) that, although the disease distribution appeared to be irregular in Wisconsin, wilted trees were found on all types of sites without apparent correlation with soil or site characteristics. In 1950, Miller (9) pointed out that the disease spreads most rapidly in dense stands of susceptible species and that the spacing of such stands within a region, and of susceptible trees within a stand, would be expected to influence the rate of disease spread. The important part that naturally occurring root-grafts could play in facilitating local spread has been reported by Kuntz and Riker (7), but there is relatively little information concerning conditions that favor root-grafting, other than the necessity for contact between the roots of closely related compatible oaks, especially those of the red oak, or Erythrobalanus group. In addition, it seems probable that at least part of the local spread can be attributed to the same agency or agencies which bring about long-distance spread.

Riker emphasized the importance of losses owing to the local intensification of oak wilt in Wisconsin (10). In this regard, recent studies made in North Carolina and West Virginia (1, 3) suggest that small groups of dead trees shown or presumed to have died from oak wilt are early results of the recently recognized presence of the disease. Some of the presumed centers of local intensification in West Virginia, which have recently produced wilting trees, include as many as 30 to 50 killed oaks and may encompass as much as an acre of land. In most instances, however, established[5] infection centers have contained only 2 to 5 dead trees when found.

In 1955, Gillespie and True (5) reported that in three northeastern counties of West Virginia (Mineral, Hampshire, and Grant) 44 percent of the 1955 season's active oak wilt infection centers included two or more currently wilting trees, and that dead oak trees were found within 50 feet of more than half of them. Therefore, local spread in these counties was an important factor in the epidemiology of the disease. In three southern counties (Kanawha, Boone and Lincoln), which included a reasonably comparable number of current oak wilt centers, only 5 percent had more than one wilting tree, and dead oaks could be found within 50 feet of the wilting trees on considerably less than half of them. Therefore, in the southern counties, long-distance spread (to trees more than 50 feet from diseased trees) appeared to

[1]Published cooperatively with the approval of the Commissioner of Agriculture as Special Survey Paper No. 8, West Virginia Department of Agriculture, and with the approval of the Director of the West Virginia Agricultural Experiment Station as Scientific Paper No. 596.

[2]Respectively, Coordinator, West Virginia Oak Wilt Survey, West Virginia Department of Agriculture, and Plant Pathologist, West Virginia Agricultural Experiment Station. The authors wish to express appreciation and thanks to Mr. Boyd Patton, State Soil Scientist, of the United States Department of Agriculture Soil Conservation Service who made available maps and other important soils data and to Mr. F. Waldo Craig, Director, Plant Pest Control Division, West Virginia Department of Agriculture for his understanding cooperation.

[3]Compatible oaks may be defined as trees belonging to the same species as, or closely related to, the diseased oaks at the infection center. Here members of the so-called red or black oak group are referred to. The term compatible is used because of the greater frequency with which the roots of different individual trees of the same or closely related species are found grafted.

[4]A disease of oak trees caused by the fungus Ceratocystis fagacearum (Bretz) Hunt.

[5]Centers which have been present for some time and contain dead trees as contrasted with initial centers where currently wilting trees are the only evidence of the presence of the disease.

be more important than local spread.

The soils in the northeastern area included many that were shallow or very shallow and some that were deep or moderately deep. Recent detailed soils maps of most other areas of the State are not available. However, most of the soils present in the southern counties are, except on the tops of ridges, deeper than the average for soils found in the northeastern counties.

The purpose of this paper is to report additional data regarding 335 oak wilt infection centers found in 1955 and 1956 in five northeastern counties on both shallow and deep soils. An attempt is made to relate soil depth, stand density of compatible oaks, and size of the infection center when initially treated to the rate of post-treatment local spread observed during the succeeding years.

METHODS AND MATERIALS

In this investigation soils described as shallow and very shallow were combined to constitute a category termed "shallow"[6]. Deep soils and soils of moderate depth were grouped together and designated as "deep"[7]. Nearly all of the soils in the study area were of medium texture. The soil types were determined by reference to pre-publication copies of recently completed soils maps sketched upon aerial photographs. These were supplied by the State office of the United States Department of Agriculture Soil Conservation Service in Morgantown. A field check on near marginal sites (constituting more than a 10 percent sampling of all sites) revealed that soil types found in the field were the same as those indicated on the soils maps. To avoid confusion, however, all sites on soils of variable depth and clearly marginal sites, as well as sites on which no compatible oaks were found within 50 feet of the diseased tree or trees, were excluded from consideration. Therefore, only 335 of the 488 oak wilt infection centers found during 1955 and 1956 in Mineral, Hampshire, Grant, Hardy and Pendleton counties were included in the study.

All of the infection centers have been revisited once and many of them two or three times annually since their initial establishment. Data on the number of currently wilting oaks, dead oaks, and live compatible oaks within the infection center at the time of initial treatment were taken from the records of the West Virginia Department of Agriculture. All infections included in this study were diagnosed as oak wilt either by experienced field personnel or on the basis of laboratory cultures. Control procedures consisted of girdling the currently wilting trees to the heartwood -- the so-called deep girdle technique (4). The average defoliation at the time of treatment was 70 percent.

Stand density was recorded in terms of number of healthy compatible oaks which were within 50 feet of the diseased tree or trees. The size of the infection center was determined by the number of currently wilting oak trees originally found and girdled plus any associated dead oaks.

RESULTS

Data given in Table 1 show that there was more local spread on shallow soils than on deep soils in each of the five counties that contributed an adequate number of infection centers to each category of comparison. In the five-county area local spread occurred from single-tree infection centers in two cases out of forty (5 percent) in deep soils, but occurred in eight cases out of 83 (9.6 percent) on shallow soils. At multiple-tree sites the comparable figures were 10 of 61 (16.4 percent) for deep soils and 41 of 157 (27.1 percent) for shallow soils. Local spread, therefore, occurred nearly twice as frequently on the shallow as on the deep soils.

The figures indicate also that the number of trees present at an infection center before treatment is closely related to the incidence of local spread after treatment. There were three times as many cases of local spread after treatment in the case of multiple-tree infection centers as for single-tree centers. This was true on both deep and shallow soils. Fur-

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[6]The shallow soils were Litz, Lehew, Calvin, Ashby, Dekalb and Vanderlip. All of these soils are less than 20 inches in depth and the shaley phases of Litz, Ashby and Calvin may be less than 10 inches deep.

[7]The deep soils were Laidig, Huntington, Monongahela, Buchanan, Brookside and Elliber. The depth of these soils exceeds 36 inches, but Buchanan and Monongahela may have a "hardpan" at 20 to 30 inches.

ther evidence of this relationship is found in Table 2 where three site-size categories were studied. For all soils, single-tree infection centers, centers including only one dead and one symptom tree, and centers with two or more dead trees or symptom trees showed local spread in 8.1, 10.6, and 30.1 percent of the cases, respectively. No repeated local spread from single-tree infection centers has as yet been observed, but 4.5 percent of the centers with one dead and one symptom tree showed repeated local spread, as did 6.8 percent of the centers consisting of two or more dead or symptom trees. The results in Table 2 also show that when divided into three groups on the basis of site size, the number of cases of initial and repeated local spread in each category is greater on the shallow than on the deeper soils.

Table 1. Relation of soil depth and size of infection centers when treated to the incidence of post-treatment local spread of oak wilt in five northeastern counties of West Virginia.

Soil type and county	Single-tree sites: Ratio of breakovers[a] to total sites	Single-tree sites: Percentage total sites broken over	Multiple-tree sites: Ratio of breakovers to total sites	Multiple-tree sites: Percentage total sites broken over
Deep Soils:				
Grant	0/3	0	0/4	0
Mineral	1/19	5.3	6/35	17.1
Pendleton	1/11	9.1	2/13	15.4
Hampshire	0/6	0	1/7	14.3
Hardy	0/1	0	1/2	50.0
Combined summary	2/40	5.0	10/61	16.4
Shallow Soils:				
Grant	2/10	20.0	6/23	26.0
Mineral	2/29	6.9	15/39	38.4
Pendleton	4/21	19.0	11/37	29.7
Hampshire	0/17	0	6/40	15.0
Hardy	0/6	0	3/12	25.0
Combined summary	8/83	9.6	41/151	27.1

[a]Breakovers are here defined as initial or repeated local spread at a known oak wilt infection center following treatment of the first tree or trees found.

The results recorded in Table 3 point to the expected relationship between the number of compatible oaks present at an infection center and the incidence of local spread from it. The data show that local spread under such conditions is directly related to the number of compatible oaks within 50 feet. However, they also show that differences in the incidence of local spread are related within each density category to the depth of the soil at the infection centers.

Therefore, it is interesting to note from Table 4 that the amount of local spread from the 488 infection centers in the northeastern counties is nearly twice as great on a percentage basis as is the rate of local spread from the 964 infection centers found elsewhere in the State. The comparatively small five-county area also provided 13 instances of repeated local spread, while in the remainder of the State only seven cases of repeated spread have been found during the ensuing years.

DISCUSSION

The three site characteristics found in this study to be directly related to the incidence of local spread after treatment are 1) depth of soil, 2) availability of compatible oaks, and 3) size of center when initially located. It seems likely that these same factors may be similarly related to incidence of local spread if no treatment is used. However, rate of local spread from untreated infection centers probably would be greater, owing to more frequent formation of oak wilt mats on non-girdled trees. From the standpoint of a control program, the most encouraging relationship to note is the relatively infrequent incidence of local spread from infection centers found and treated while they were single-tree sites. More alarming is the fact that the infection centers which had built up into multiple-tree sites before they were found showed a much higher percentage of local spread, and several even showed repeated local spread in the 2 to 3 year period that has followed their discovery. This suggests an

Table 2. Relation of soil depth and size of infection centers when treated to the incidence of post-treatment local spread and repeated local spread of oak wilt in five northeastern counties of West Virginia.

Site size	Ratio of breakovers to total sites			Percentage of total sites with breakovers			Ratio of repeat breakovers[a] to total sites			Percentage total sites repeat breakovers		
	Deep soil	Shallow soil	All soils	Deep soil	Shallow soil	All soils	Deep soil	Shallow soil	All soils	Deep soil	Shallow soil	All soils
Single tree sites	2/40	8/83	10/123	5.0	9.6	8.1	0/40	0/83	0/123	0	0	0
Sites with one dead[b] and one symptom tree	1/21	6/45	7/66	4.8	13.3	10.6	0/21	3/45	3/66	0	6.7	4.5
Sites with two or more dead plus symptom trees	9/40	35/106	44/146	22.5	33.0	30.1	1/40	9/106	10/146	2.5	8.5	6.8
All sites	12/101	49/234	61/335	11.9	20.9	18.2	1/101	12/234	13/335	1.0	5.1	3.9

[a]Repeat breakovers are here defined as cases in which annual local spread had occurred for 2 or more years after treatment of the first tree or trees found. The wilting trees located in succeeding years are treated when found.
[b]Dead oaks, especially two or more found in association with currently wilting trees, are presumed to have died of oak wilt (1, 3).

Table 3. Relation of soil depth and of live compatible[a] oaks found within treated oak wilt infection centers to the incidence of post-treatment local spread of the disease in five northeastern counties of West Virginia.

Number of trees	Ratio of breakovers to total sites			Percentage of total sites with breakovers			Ratio of repeat breakovers to total sites			Percentage total sites repeat breakovers		
	Deep soil	Shallow soil	All soils	Deep soil	Shallow soil	All soils	Deep soil	Shallow soil	All soils	Deep soil	Shallow soil	All soils
Up to 6 live compatible oaks	8/69	27/148	35/217	11.6	18.2	16.1	1/69	6/148	7/217	1.4	4.0	3.2
From 7 to 12 live compatible oaks	3/25	14/69	17/94	12.0	20.3	18.1	0/25	3/69	3/94	0	4.3	3.2
From 13 to 20 live compatible oaks	0/7	9/17	9/24	0	52.9	37.5	0/7	3/17	3/24	0	17.6	12.5

[a]Compatible oaks may be defined as trees belonging to the same species as, or closely related to, the diseased oaks at the infection center. Here members of the so-called red or black oak group are referred to. The term compatible is used because of the greater frequency with which the roots of different individual trees of the same or closely related species are found grafted.

Table 4. Incidence of post-treatment local spread and of repeated local spread in five northeastern counties of West Virginia as contrasted with incidence of comparable spread elsewhere in the State.

Area	Total number infection centers 1955-1956	Original breakovers to date	Percentage breakovers of total sites	Number of repeat breakovers	Percentage repeat breakover of total sites
Five northeastern counties	488[a]	81	16.6	13	2.7
All other counties	964	76	7.9	7	0.7

[a]This figure includes the 335 infection centers which comprise this study, together with 153 additional infection centers found in the same counties during 1955 and 1956 but excluded from the study either because they had no compatible oaks within 50 feet or because the soil types on which they were situated were of variable depths or because they were situated too close to the boundaries between soil types to be safely assigned to any.

urgent need to find the infection centers and treat them before they expand into multiple-tree sites.

In West Virginia, the results show that in the northeastern counties local spread of oak wilt represents a critical situation.

More generally speaking, it may be that in the shallow and very shallow soils which characterize much of the study area, but are seldom found extensively elsewhere in the State, there may be a crowding of tree root systems so that root grafts occur more frequently. Some of these shallow soils are also stony, even at or near the surface, which further restricts the soil space available for tree roots; conceivably this could be a factor favoring the more frequent occurrence of root contacts between trees, leading to root grafts and to root-graft transmission of oak wilt. Another possibility is that shallow-rooted trees might be more subject to attack by bark and wood-boring beetles which could occasionally serve as vectors (2, 11), especially during dry summers. This suggests the possibility that in certain Appalachian soils the incidence of overland spread as well as root-graft spread is related to soil depth.

The deep-girdle apparently reduces rate of overland spread by curtailing the longevity of the fungus above the girdle and by suppressing fungus mat formation. However, this treatment probably does not prevent or significantly reduce the incidence of root-graft spread, since cultures from trunk samples taken from the lower bole of currently diseased trees that are more than 50 percent defoliated seldom fail to yield the fungus.

It seems that in West Virginia, as in many parts of the midwestern oak wilt area, factors are present which result in wide variations in the rate of local disease intensification. This article merely points to the importance of the three site characteristics investigated, all of which were found to be directly correlated with the rate of local spread in the study area. Just how these factors bring about the results observed is not yet determined. The results will be useful, however, in planning the different phases of future research and of control programs in West Virginia.

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WEST VIRGINIA DEPARTMENT OF AGRICULTURE, AND WEST VIRGINIA AGRICULTURAL EXPERIMENT STATION

BRIEF NOTES ON PLANT DISEASE OCCURRENCE

A NEW HOST FOR THE LARCH DWARFMISTLETOE

By Donald P. Graham[1]

The larch dwarfmistletoe, Arceuthobium campylopodum Engelm. f. laricis (Piper) Gill, common on western larch (Larix occidentalis Nutt.) and subalpine larch (L. lyallii Parl.), has recently been found on three planted jack pines (Pinus banksiana) on the Priest River Experimental Forest in northern Idaho. This species of dwarfmistletoe had been reported previously in nature on lodgepole pine, western white pine, Engelmann spruce, and subalpine fir[2]. However, this is believed to be the first report of its occurrence on jack pine. A. americanum Engelm., the dwarfmistletoe that usually attacks jack pine, was not found in the area.

The infected jack pines were planted 25 years ago and are about 25 feet high. They are located on the administrative grounds of the experimental forest under several heavily infected western larch overstory trees. The morphology and vigor of the dwarfmistletoe shoots produced on the jack pine are characteristic of the parasite on its usual host tree. Witches'-brooms, similar to those that form on western larch, are also present on the infected jack pine trees.

Because the natural botanical range of western larch and jack pine do not overlap, this crossover has no importance in forest management. However, the fact that jack pine is susceptible to the larch dwarfmistletoe is important for future studies of host-parasite relationships and in clarification of the taxonomy of the genus Arceuthobium.

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[1]Pathologist, Intermountain Forest and Range Experiment Station, Forest Service, United States Department of Agriculture, Ogden, Utah; stationed at Inland Empire Research Center, Spokane, Washington.

[2]Kuijt, Job. 1954. Some notes on the larch mistletoe in British Columbia. Canad. Dept. Agr. Forest Biol. Div., Bi-monthly Prog. Rept. 10 (6).

A LATENT VIRUS OF HOPS DETECTED BY CUCUMBER INOCULATION[1]

By P. R. Fridlund

During tree fruit virus investigations begun in 1956, a latent virus was found in hops, Humulus lupulus. The virus was transmissible to cucumber by juice inoculation after the method of Boyle et al.[2]. To the writer's knowledge no hop virus had previously been reported to be transmissible to an annual plant.

The virus transmits readily from hops to cucumber, in which it causes symptoms indistinguishable from certain isolates of the necrotic ring spot virus of Prunus. In limited trials it was also transmissible from hop to hop by both approach grafting and the above juice inoculation technique. Inoculations from cucumber to 35 other herbaceous plant types caused no symptoms nor was any virus found when the inoculated plants were reindexed into cucumber. Juice inoculations from cucumber and hop to Prunus mahaleb were negative. Approach-grafting results from hop to Prunus tomentosa were also negative, as were results from budding stem bark patches from diseased hops to Shiro-fugen flowering cherry and P. tomentosa.

The identity of the virus is not known, as studies in hops have not been made. These limited studies were made only to determine if it also was a virus of Prunus. Apparently it is not. The principal purpose of this report is to call attention to the existence of a latent virus in hops which is readily and conveniently detected by transmission to cucumber.

IRRIGATION EXPERIMENT STATION, WASHINGTON AGRICULTURAL EXPERIMENT STATIONS, PROSSER, WASHINGTON

[1]Irrigation Experiment Station, Washington Agr. Exp. Stas., Prosser, Washington. This work was conducted under Project No. 1262 and was supported by Inter-regional research project IR-2.

[2]Boyle, J. S., J. Duain Moore, and G. W. Keitt. 1954. Cucumber as a plant host in stone fruit virus research. Phytopathology 44: 303-312.

NEW RECORDS OF PLANT DISEASES IN NEW MEXICO

By C. H. Hsi

Stripe rust of wheat was recorded for the first time in New Mexico. Among the hard red winter wheats grown in the area, Wichita, Tenmarq, Westar and Concho were observed to be susceptible to the disease, whereas Turkey, Cheyenne and Aztec were highly resistant. Source of infection was wind-blown spores from the east. Heavy and moderate precipitation in March and April 1958, respectively, and much below normal temperatures in those 2 months were probably conducive to the establishment and subsequent development of stripe rust on susceptible wheat plants grown in the area.

Head smut of sorghum was also recorded for the first time in New Mexico. Considerable head smut was found in fields or rows planted with RS 610, Amak R 10 and Combine 7078. Sources of original inoculum were believed to be seed-borne, although wind-borne spores from Texas also could be a source of infection. Head smut will be a potential threat to the area sorghum production once it is established in the soil, as many of the commonly grown hybrids and varieties are susceptible to the disease at the present time.

PLAINS SUBSTATION, NEW MEXICO AGRICULTURAL EXPERIMENT STATION, NEW MEXICO STATE UNIVERSITY, CLOVIS

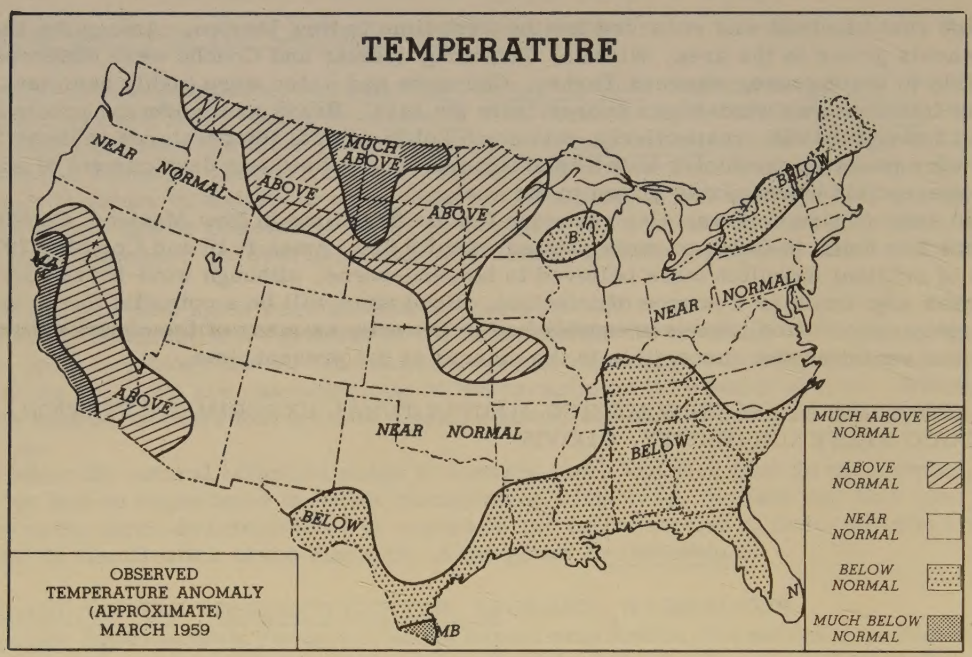
ANNOUNCEMENT

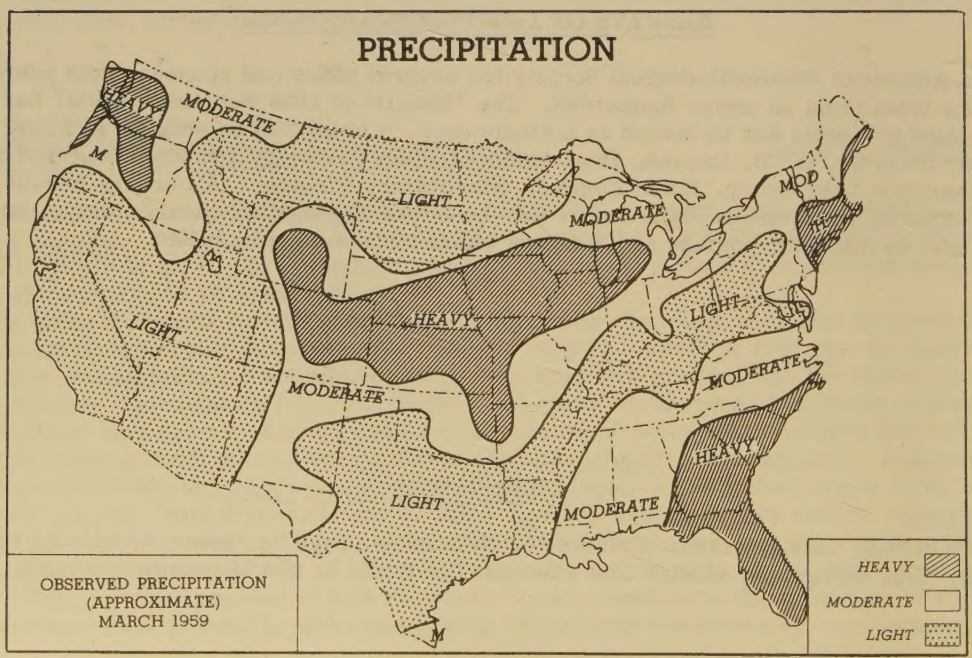
RESULTS OF 1958 FUNGICIDE TESTS

The American Phytopathological Society for several years has sponsored the publication of results from tests on newer fungicides. The "Results of 1958 Fungicide Tests" has again been printed privately and is issued as a single copy. Copies can be secured at $1.00 per copy only from Dr. A. B. Groves, Department of Plant Pathology and Physiology, Winchester Fruit Research Laboratory, Rural Route 3, Winchester, Virginia. All orders should be accompanied by remittances made out to The American Phytopathological Society. An added charge will be made for postage and handling where orders must be billed.

CORRECTION

REPORTER, January issue (Volume 43, Number 1), page 9: Under Materials and Methods, first paragraph, change line 4 to read "of 2 1/2, 5, and 20 pounds per acre..."





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.